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Southern Medical Association, Thirty-First Annual Meeting, New Orleans, Louisiana, November 30-December 1-2-3, 1937

TABLE OF CONTENTS

The Roentgen Treatment of Infections. Willis F. Manges, Philadelphia, Pennsylvania 243

The Bone Changes in Sickle Cell Anemia. L. W. Diggs, H. N. Pulliam and J. C. King, Memphis, Tennessee 249

Discussed by Maxwell M. Wintrobe, Baltimore, Md.; V. P. Sydenstricker, Augusta, Ga.

Thyroidectomy: Factors Influencing the Mortality. Daniel C. Elkin, Atlanta, Georgia 260

Diverticulum of the Urinary Bladder. Neil S. Moore, St. Louis, Missouri 263

Discussed by James L. Estes, Tampa, Fla.

Recent Developments in the Cinematography of the Fluoroscopic Image. William H. Stewart and F. H. Ghiselin, New York, New York 268

Lupus Vulgaris in the Far South. Elmo D. French and Rothwell Lefholz, Miami, Florida 270

Discussed by J. Lee Kirby-Smith, Jacksonville, Fla.; William F. Spiller, Galveston, Tex.

Suggestions and Inquiries on the Therapy of Skin Diseases. M. Toulmin Gaines, Mobile Alabama 278

Discussed by Emmett R. Hall, Memphis, Tenn.

Continued on Next Page

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TABLE OF CONTENTS

The Mass Control of Syphilis. R. A. Vonderlehr and L. J. Usilton, Washington, District of Columbia 281

Discussed by Dudley C. Smith, University, Va.

Morbid Anatomic Changes Following Artificial Fever, With Report of Autopsies. E. Lloyd Wilbur and Joseph B. Stevens, Durham, North Carolina 286

Discussed by Walter M. Simpson, Dayton, Ohio; Harry C. Schmeisser, Memphis, Tenn.

A Psychiatrist's View of the Problem of Prevention in Pediatrics. James S. Plant, Newark, New Jersey 290

Maternal Mortality in Georgia During the Year 1935. E. D. Colvin, Atlanta, Georgia 292

A Study of the Vaginal Flora in the Normal Female. Bayard Carter and C. P. Jones, Durham, North Carolina 298

Discussed by Richard W. TeLinde, Baltimore, Md.; Karl John Karnaky, Houston, Tex.

The Prevention of Deaths from Measles. Huntington Williams, Baltimore, Maryland 304

Adrenal Hemorrhage in Children. Warfield M. Firor, Baltimore, Maryland 306

The Present Status of Flexible Tube Gastroscopy. James L. Borland, Jacksonville, Florida 310

Discussed by Rudolph F. Schindler, Chicago, Ill.; E. B. Freeman, Baltimore, Md.; Porter P. Vinson, Richmond, Va.

Carcinoma of the Gallbladder: Studies of 24 Cases in Georgia. Robert L. Rhodes and Robert B. Greenblatt, Augusta, Georgia 315

Discussed by Frank S. Lynn, Baltimore, Md.

Fracture Service in a Small General Hospital. William T. Hammond, Easton, Maryland 318

Discussed by H. Earle Conwell, Birmingham, Ala.; Robert W. Johnson, Jr., Baltimore, Md.

Acute Infections of the Middle Ear and the Mastoid Cells. John J. Shea, Memphis, Tennessee 322

Discussed by Edward A. Looper, Baltimore, Md.

Microscopic Anatomy of the Temporal Bone Showing Pathologic Routes of Extension to the Cranial Cavity. Dorothy Wolff, St. Louis, Missouri 326

Discussed by S. J. Crowe, Baltimore, Md.; Ella M. A. Enlows, Washington, D. C.

Concerning Norms, Nebulae and Foreign Bodies in Requirements for Clinical Certifications. George H. Meeker, Philadelphia, Pennsylvania 329

Diseases of the Nervous System Caused by Nutritional Deficiency. Erwin Wexberg, New Orleans, Louisiana ... 334

Discussed by Titus H. Harris, Galveston, Tex.

Dietary Deficiencies as a Cause of Anemia. Paul W. Clough, Baltimore, Maryland 342

EDITORIALS 346

X-Ray: Further Progress
Posterior Pituitary Extracts in Eclampsia
Gleanings from Recent Journals
Twenty-Five Years Ago

CORRESPONDENCE 349

An Efficient and Inexpensive Skin Cleansing Formula. Wallace Marshall, University, Alabama.

BOOK REVIEWS 349

SOUTHERN MEDICAL NEWS 354

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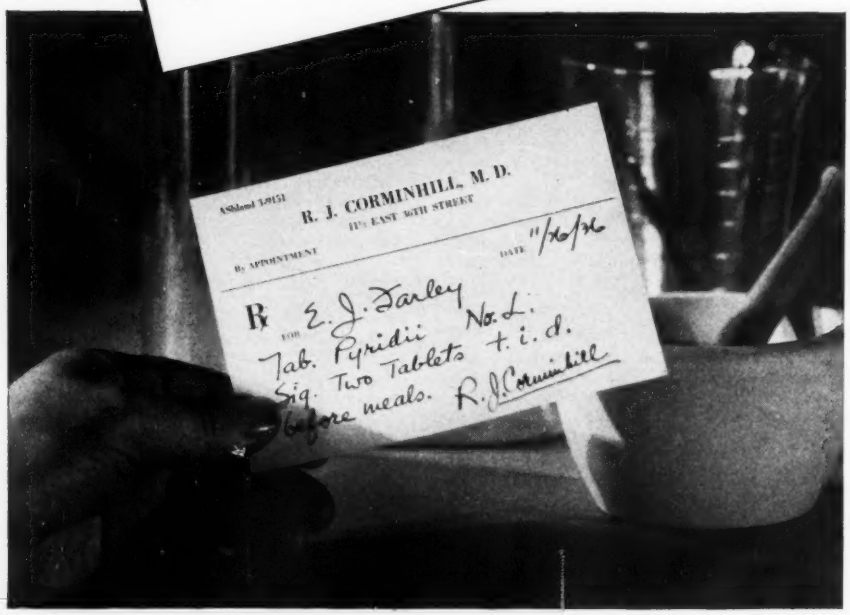

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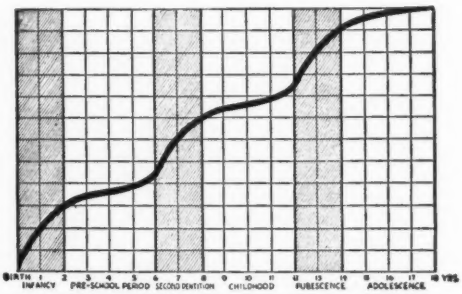

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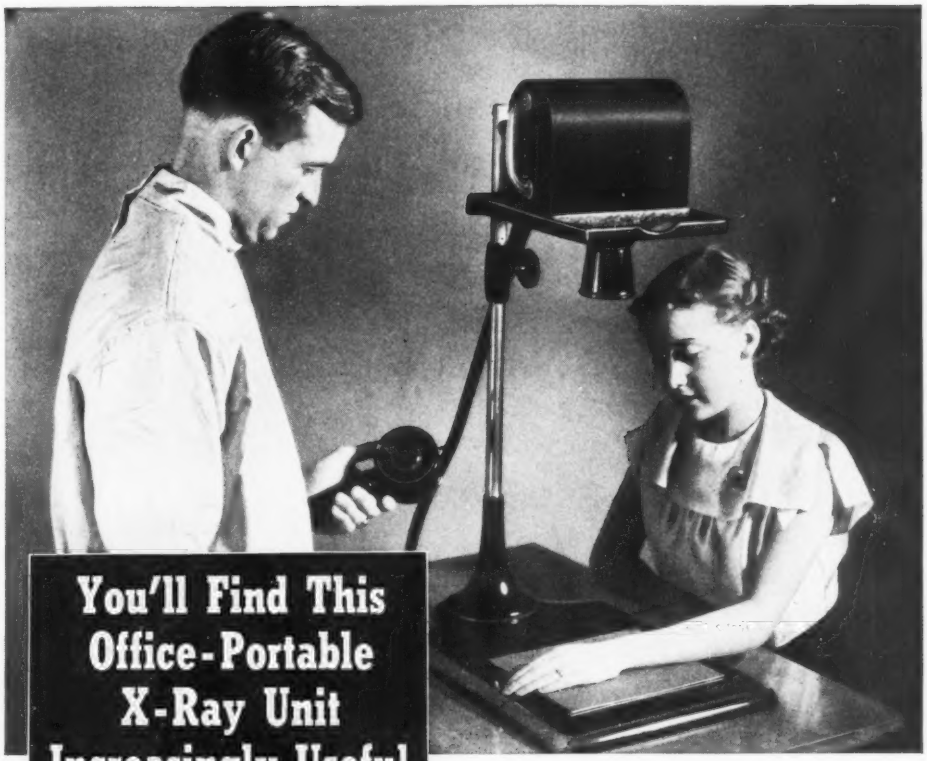

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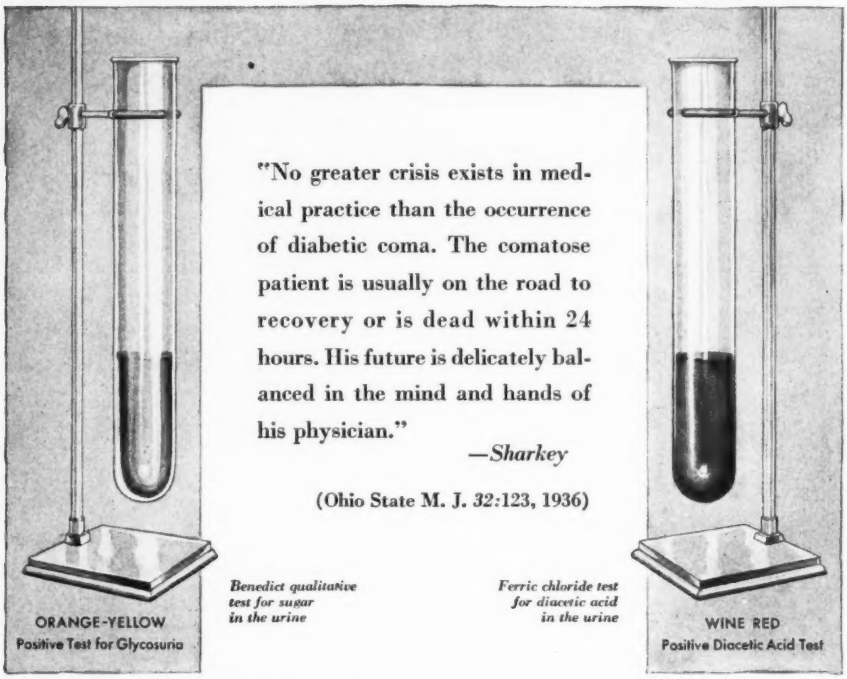
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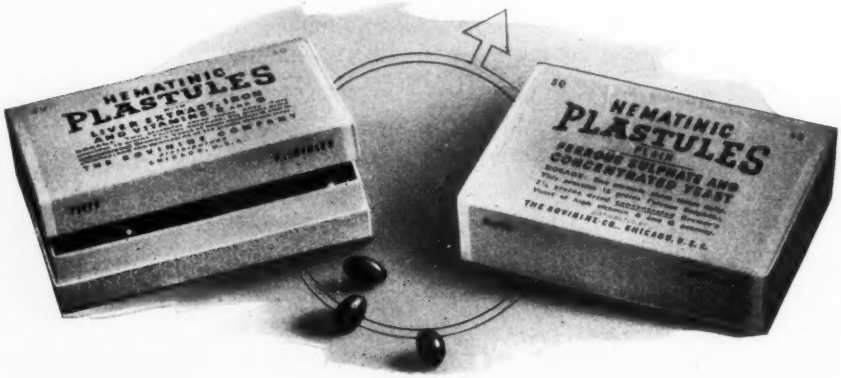

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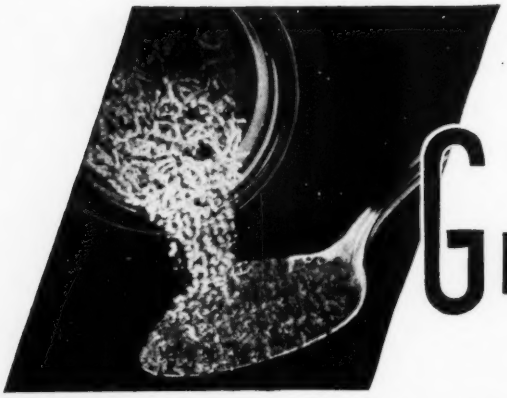

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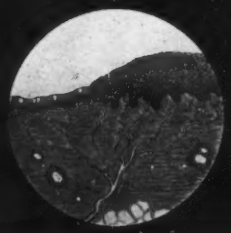

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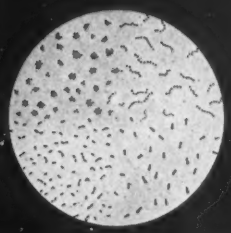

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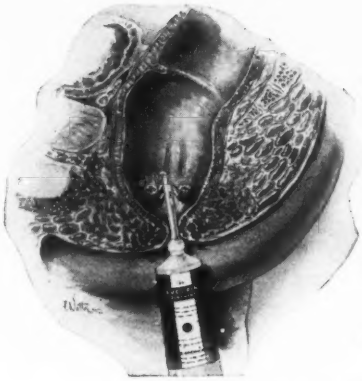

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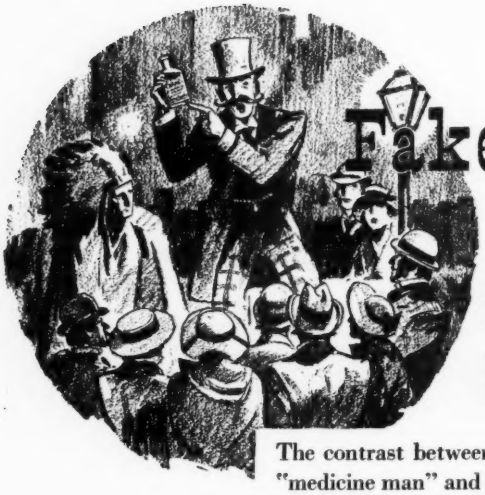

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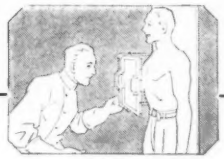

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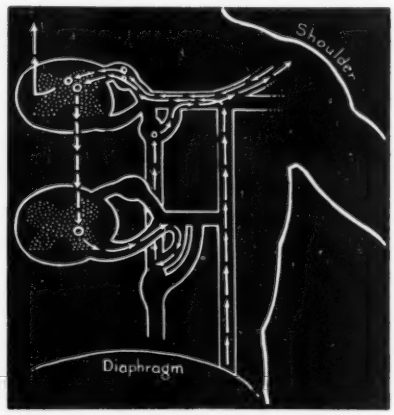

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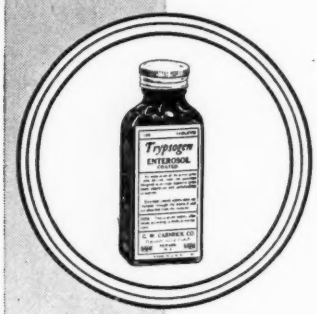

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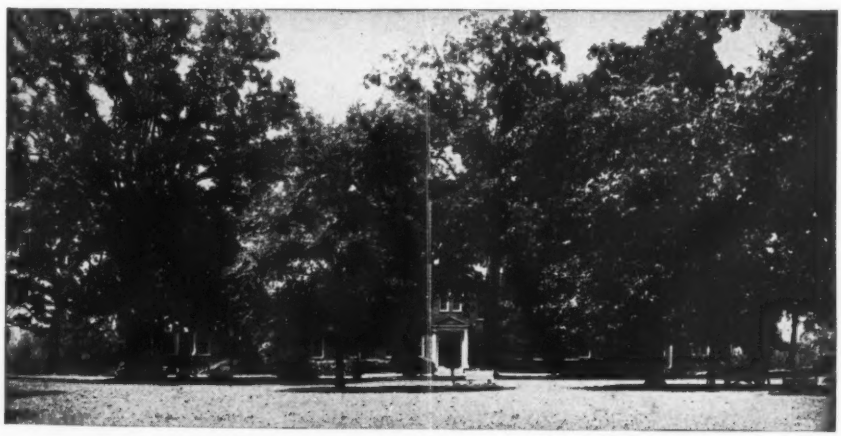

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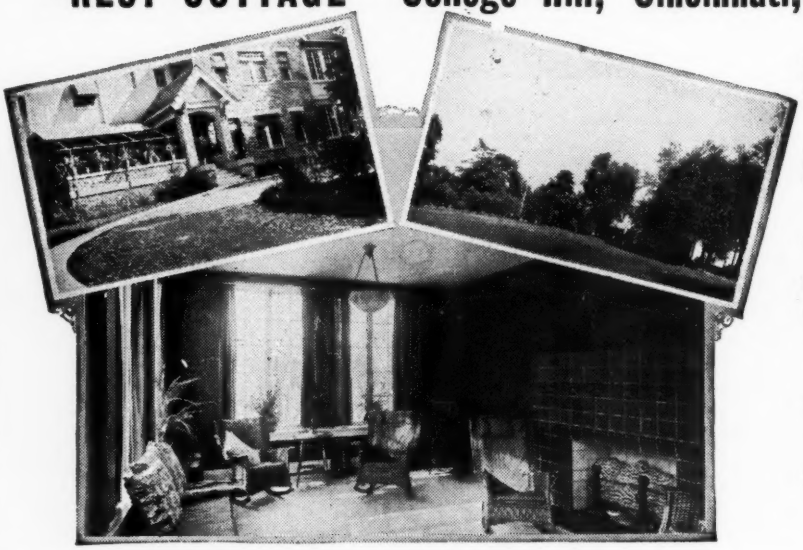

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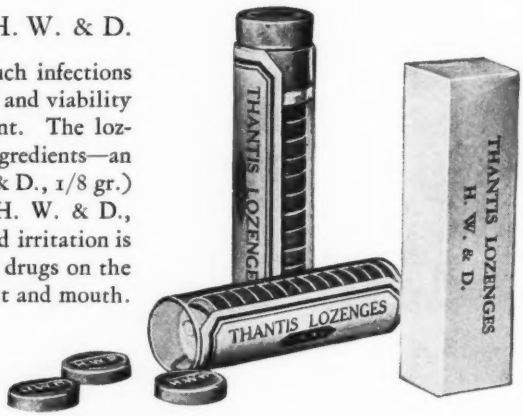

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Volume 30 MARCH 1937 Number 3

THE ROENTGEN TREATMENT OF INFECTIONS*

By WILLIS F. MANGES, M.D.†
Philadelphia, Pennsylvania

Roentgen ray therapy in malignant growths, certain benign tumors, Hodgkin's disease, leukemia, and in many of the diseases of the skin is a generally accepted procedure in the medical profession throughout the world. Large sums of money have been spent in research and in the improvement of apparatus with the hope that the small percentage of cures may be increased or that the period of palliation may be extended. While much has been accomplished, the results up to the present are by no means satisfactory.

If the results of roentgen therapy in malignancy could in any way compare with the results of only small doses of roentgen rays in the treatment of acute infections and inflammation, there would be relatively little concern about cancer or sarcoma. Does it not seem strange, then, that while all roentgenologists, and I may say all surgeons, have given much thought and energy to a difficult problem, they have paid but little attention to an almost equally important and much less difficult problem?

One need only search the American literature to realize how little the surgeon knows or recognizes its value, and likewise the literature shows that relatively few roentgenologists have taken any interest in the subject. As a matter of fact, Dr. Hodges, your worthy President, has probably done more and written more on this subject than any other American roentgenologist, and you would profit well by reading his articles.

It is true that a great deal has been done in European countries, especially in Germany, and nearly all of the research work bearing on the subject has been done abroad. The main responsibility for neglect in this country must in fairness be placed on the roentgenologist. Certainly, I have encountered few surgeons who have not welcomed help when I have suggested it, and numbers of them have been the grateful recipients.

I have decided to discuss the subject from a general viewpoint rather than to report a special group or an individual type of case, because you represent all of the specialties, as well as general medicine and surgery. In order that I may not be misunderstood, the acute infectious diseases of childhood are not to be considered a part of the discussion. It is worthy of note, however, that Hickey,[5] in 1922, proved that diphtheria carriers could, in a large percentage of cases, be rid of the germs by roentgen ray treatment over the upper respiratory tract.

I would also exclude those infections for which a definite immunizing agent or a specific vaccine or antitoxin has been found, such as tetanus and smallpox.

I do not advocate roentgen ray treatment for pulmonary tuberculosis, but it certainly is the method of choice in early tuberculous adenitis,[17 19] and is a helpful adjunct in tuberculous peritonitis. With very few exceptions, it may be said that it is of real value in all other sorts of infections.[10 13 14]

It might be well to mention a few of the more common clinical conditions in which the results under reasonably favorable circumstances are outstanding. Such a list would include: furuncles,[4 6] carbuncles, particularly carbuncle of the face,[1] paronychia, felon, abscess anywhere, erysipelas,[9] cellulitis,[7] lymphangitis, pelvic infection in the female, cystitis, periproctitis, parotitis,[8] acute sinus and mastoid infection, gas bacillus infection[12] (gas gangrene), infection from human bites,[15] postoperative[11] wound inflammations and infections, unresolved pneumonia, and even lobar pneumonia[18] if treated in the early

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†Dr. Manges died November 24, 1936.

stages. The optimum time for the treatment is when the first definite evidence of the infection is established, with pain, swelling, redness, tenderness, fever, leukocytosis, lymphangitis, adenitis, or other indications that natural forces are in a process of reaction against the invasion. In other words, the inflammatory process must be established before the radiation is effective.

When treated in this early stage, a large percentage of the suppurative lesions disappear without suppurating, pulmonary consolidations resolve early, lymphangitis and lymphadenitis subside spontaneously, temperature falls by crisis or rapid lysis, pain, though it may be increased for a few hours, abates speedily, and other signs go more gradually. One of the remarkable clinical evidences is that many of the most toxic patients have early a real sense of relief, regardless of change in temperature. Certain lesions, such as erysipelas or metastasizing infections, are apt to advance to adjacent or distant parts even when treated early, but prompt attack on these new areas usually brings about the desired result. Treating an adjacent uninvolved area will not prevent the spread of erysipelas.

Now, if the treatment is not given in the early stage in localized, suppurative processes, it becomes the most helpful of all adjuncts to surgery. Even if suppuration has taken place, it is quite profitable to give radiation prior to incision and wait until the lesion becomes more localized and superficial, in just the same way that you have always applied heat or poultices and waited until the abscess has come to a head. In fact, radiation should entirely replace poulticing in any form.

These rules apply equally to the case in which an infection which has been established, incised, and draining, shows a tendency to spread. Instead of applying heat, radiation should be used. This is particularly true of the infection that comes from a needle puncture of a finger of the nurse or surgeon. A little radiation will save a phalanx, finger, hand, arm, or life, depending upon the stage at which the radiation is given; and the relief from pain is almost invariably more complete than with full doses of morphine.

It is also of real value in the more advanced stages of suppurative processes, such as the palmar abscess which has drained pus for weeks, or the deep-seated pelvic infection that defies the effort of the gynecologist, or the diffuse suppurative pneumonitis that does not form a true abscess, and makes it difficult for the surgeon or bronchoscopist to obtain adequate drainage.

Under such conditions radiation, in addition to combating the infection, changes the character of the dammed-up exudate, making it more liquid, hence more responsive to methods of drainage.

The rules governing this treatment are clear, concise, and vary only slightly, regardless of the lesion, its stage of development, or location. The treatment is limited to the area of the disease, but the part may be treated on opposite sides, as, for example, in the hand the palmar aspect may be treated one day and the dorsal aspect the next day. In the acute stages always, and with few exceptions in the later stages of superficial infections, the dose[3] to a given area should be no more than from 10 to 20 per cent of an erythema dose, or, in terms of units, from 60 to 120 R, relatively low voltage, from 80 to 135 kilovolts, and filtration of from 1 to 4 mm. of aluminum are preferable. For example, at the Jefferson Hospital we treat all erysipelas cases at the bedside with portable apparatus, operating at 80 kilovolts, through 1 mm. of aluminum; and such lesions as felons, superficial carbuncles, boils, cellulitis, and so forth, with the machine operating at 135 kilovolts, through 4 mm. of aluminum.

For deep-seated and more massive lesions, such as pelvic infections, we use up to 200 kilovolts, with a filter of ½ mm. copper and 1 mm. of aluminum, but the dose in terms of R units is the same, that is, from 60 to 120, in all but exceptional cases.

Such a dose may be repeated[2] a few times at intervals of one, two or three days, or as much as a week. The superficial lesion rarely requires more than one to three treatments.

The exceptions to these rules, as pointed out by Dr. Hodges, are certain processes, such as chronic suppurative parotitis, and especially infectious granuloma.

You can readily see that there is nothing harmful in the procedure.

And now I must say with emphasis that all other methods or aids in the treatment of infection are to be called upon when indicated, with the one exception that strong, moist heat or poultices should not be used in cases that are treated with x-ray. When pus is present, it must be drained surgically, and stimulation and other supportive measures must not be neg-

lected. Rest and elevation also are important, and severe anemia must be controlled.

The explanation of the effect of radiation in these conditions is not yet entirely satisfactory, but the more recent research work[16] tends to show that phagocytosis is increased, that nonspecific antibodies are formed, that the known antibody formation is enhanced, that ferments and enzymes are developed, that in the case of gas bacillus infections hydrogen peroxide is liberated in the infected areas, and that the pain is relieved because of certain chemical changes, and relief of tension.

The clinical data are of far greater importance up to the present than the experimental, particularly because comparatively few types of infection have been studied experimentally. Most of the research work has been done in small animals with large doses of radiation to the entire body, and in many instances the radiation has been given prior to the infection, whereas we treat only the involved area, use small doses, and only after the inflammation or infection is definitely established.

It would lead me too far afield if I were to attempt to review the literature, but you will find in the published paper a few references that may be of interest to you.

I should like to present a few cases to illustrate and emphasize the general discussion of the subject.

Case 1.—R. McL., 25, was admitted May 24, 1935. During the night before admission he struck another man in the mouth, inflicting a deep wound on his right hand. He applied to the accident room for treatment and because of the incorrect history he gave, the wound was merely cleansed and sutured. The following morning his hand was markedly swollen, there were red streaks of lymphangitis up the arm, and the axillary lymph nodes were swollen and tender. He was given a treatment on admission. His temperature on entering the hospital May 24 was 100°. The succeeding day it was normal and remained so until his dismissal. A roentgenogram of the hand revealed the presence of a portion of a tooth. The sutures were removed and later the same day the fragment of tooth was removed. He was discharged well on May 31. Suppuration did not occur.

This case represents that group of infections from human bites. It seems reasonable to believe that the treatment was extremely efficacious, in view of the rapid development of lymphangitis and lymphadenitis, as well as the early and severe reaction in the hand.

Case 2.—Miss K., a nurse, aged 23, on March 10, 1936, jagged her right thumb with a needle. Infection followed and on the tenth day there was suppuration which was drained surgically. A roentgenogram showed partial destruction of the distal phalanx. She suffered great pain and had lost eight pounds in weight. She was given 75 R on March 20, and that night slept without sedative. She had another similar treatment on March 22, after which the lesion gradually healed without further destruction of bone or soft tissue.

Case 3.—Dr. W. K., a surgeon, injured his right index finger with a pair of tissue forceps on July 30, 1928. He

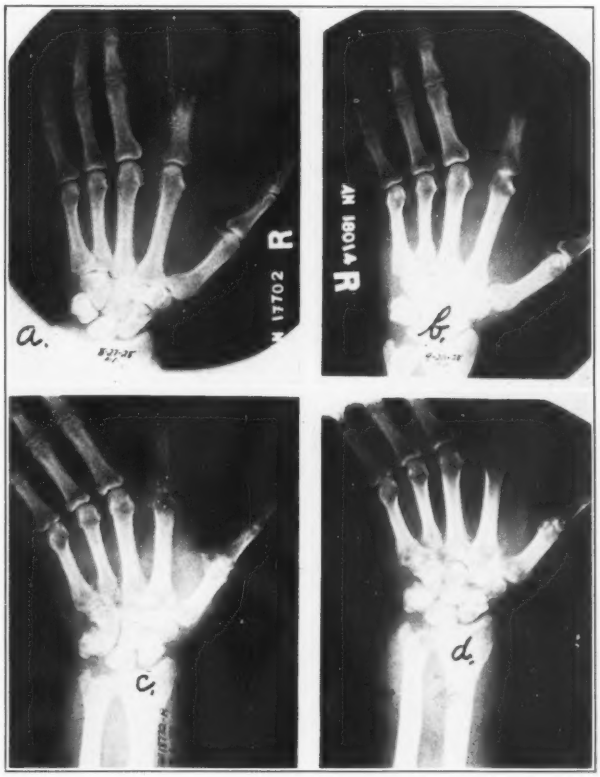

Fig. 1, Case 3

Dr. W. K. Osteomyelitis. (a) Twenty-seven days after injury. Note absence of two phalanges; slight defect, base of proximal phalanx; no joint destruction. (b) Twenty-four days later. Note increased bone destruction and destruction of articular cartilage. (c) Thirty-nine days later. Increased bone absorption of finger; beginning decalcification, carpal bones. Note beginning swelling in the soft parts of the forearm. (d) Eight days later. Note increased decalcification; increased swelling, forearm. First x-ray treatment given on this day.

had a throbbing pain that evening, and on the following morning he had a white cell count of 24,000. The finger was incised through the point of injury on July 31. No pus was obtained. Culture showed *Streptococcus hemolyticus*. Another incision and drainage were instituted on August 18.

The distal and middle phalanges were removed later, and incisions were made in the palm of the hand.

On October 18, the hand was again incised widely. This gave a great deal of relief. Later the forearm required incision and drainage.

On November 14, he came to my office for a roentgenographic examination of the hand and forearm. The cartilage of the carpometacarpal joint was destroyed and there was partial destruction of the proximal phalanx. The incisions were still draining pus.

I asked him whether he thought his surgeon would object to my giving him a roentgen ray treatment, and when he said it would be all right, I gave him 100 R over the hand and forearm, after which he had his first comfortable night. This dose was repeated on December 3, December 26, and January 16, 1929. Improvement was progressive, the metacarpophalangeal articulation ankylosed, and the destroyed area of the phalanx healed. Later the proximal phalanx and the head of the metacarpal were removed because they interfered with his work.

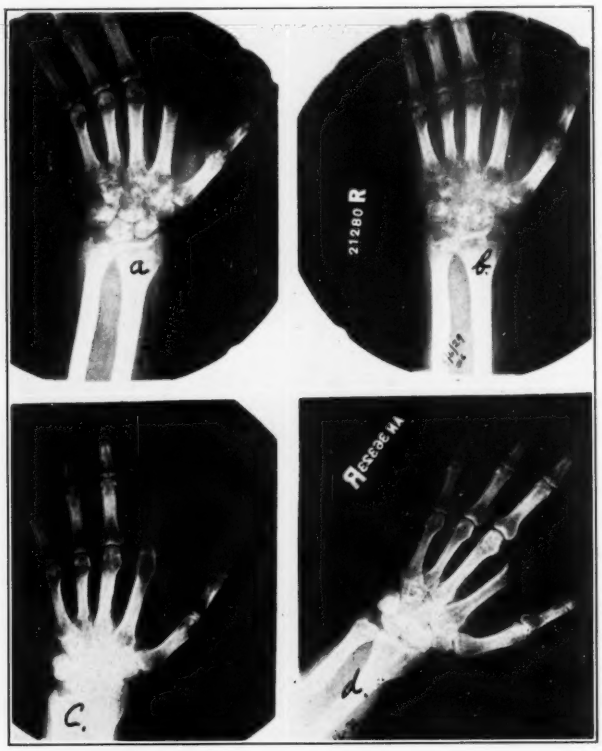

Fig. 2, Case 3

Dr. W. K. Osteomyelitis. (a) Forty-two days later. Swelling in forearm has decreased; decalcification of carpal bones has increased. (b) Six weeks later. Forearm swelling has disappeared. Note beginning bone repair at metacarpophalangeal joint. (c) Six months later. Ankylosis at metacarpophalangeal joint. Normal density of carpal bones. (d) Shows metacarpal head and phalanx removed to make the hand more useful.

This case illustrates the frequent results of surgery on infections. I firmly believe that if instead of the very first incision he had been given roentgen ray treatment, the finger or hand never would have required incision.

Case 4.—J. M., aged 43, developed an infection in the nose. It was quite tender and painful. Seventy-five R gave marked relief and 35 R on three subsequent days caused the lesion to disappear completely.

Case 5.—P. G., aged 55, developed an infection of the lower lip and was given 5 doses of 75 R on alternate days. The lesion ruptured spontaneously and healed without surgical interference.

Case 6.—T. P., aged 36, had a furuncle on the cheek. Suppuration was evident, but instead of incision, the lesion was given 75 R three times. Relief from pain was very prompt, the lesion ruptured spontaneously, and then healed promptly.

Case 7.—G. McK., aged 28, admitted Dec. 5, 1935, had had frequent "boils" in the previous years, usually on the back of the neck. Three days before admission he began to develop a "boil" on the left cheek, on which he used ichthyol. He noticed a little pustule the night before admission and squeezed it. By morning the entire left face was enormously swollen and tender. On admission he had a leukocytic count of 31,000, his temperature was almost 102°, and he was quite toxic. He was given 75 R with a portable apparatus at his bedside the same day; this was repeated the next day, and on two subsequent days he had small doses. His leukocyte count was 12,800 the day following his first treatment, and there was no pain after the first 24 hours. His temperature was normal two days after admission. Surgical interference was not necessary.

Case 8.—B. C., aged 23, had an infected upper lip and face. Eight days previous to admission he squeezed a pimple near his upper lip. The upper lip and face were badly swollen and discolored, he was delirious, and died in 48 hours.

I have no doubt that this patient would have recovered if he could have been treated during the first 48 hours. His history was exactly the same as that of the previous case.

Case 9.—Wm. D., aged 27, was admitted Dec. 22, 1935, with the diagnosis of erysipelas and rheumatic heart disease. He retired at 9:00 p. m., December 20,

feeling well. The next morning at 3:00 o'clock he was awakened by discomfort in his nose and noticed that the nose was very red and swollen. The next day he was sent to the hospital with the face greatly swollen. He was given 75 R on the day of admission and 50 R the following day.

Dr. Knowles, Professor of Dermatology, gave a grave prognosis because the patient was an alcoholic, but he proceeded to improve and was discharged well of his erysipelas on January 6. His temperature on the day of admission to the hospital was 102°. It remained at approximately that level for two days, then gradually fell. In six days it was normal. The pulse rate followed approximately the same line. The day after admission it was 150. Five days later it was normal.

Case 10.—M. E., aged 58, was admitted on November 16, 1934, with a diagnosis of chronic myocarditis, auricular fibrillation and questionable encephalitis. The chief complaints were lethargy, weakness and dyspnea.

Five weeks before admission he became very weak, dyspneic and distended. On admission he was markedly confused mentally. This increased and he became so stuporous that he could be aroused only with difficulty. On December 17, it was noted that he was in a comatose condition. He lingered in this way and on January 2 he developed erysipelas on the nose and the right side of the face. His temperature on that day was 102° and it remained at that level for nine days.

The first roentgen ray treatment was given on January 4. He was treated again on January 5 and 7. His temperature was normal on January 13. Continuation of the temperature after the erysipelas disappeared was probably due to the original encephalitis.

The erysipelas cleared up promptly, and on February 8 he was discharged mentally clear, but with fibrillation still present.

I am not prepared to estimate the effect of the roentgen rays on the cause of the encephalitis; but at least it did no harm, and I feel sure that, in view of the progressive nature of the illness prior to the treatment, it is reasonable to assume that either the erysipelas or the roentgen rays played an important part in his recovery from encephalitis.

Case 11.—R. S., aged 20, was admitted May 20, 1936, with a diagnosis of pneumonia. The onset was not typical. She had cough, loss of appetite for one week, weakness and dyspnea for three days, pain in the chest one day. The temperature was 102.6° on admission, and it reached 102° every day through May 24. The pulse rate through the same period remained close to 120°. May 21 pain started in the right side. On May 23 she coughed up large quantities of thick, greenish sputum.

On May 25 roentgenograms showed consolidation and partial atelectasis of the left lower lobe and partial consolidation of the right lower lobe. The temperature was normal on May 26 and subsequently did not rise above 99°. On May 27, 108 R were given over the lower chest posteriorly. A similar dose was given on May 29, June 1, 6, 9 and 25, treating alternately anterior and posterior areas. The temperature dropped rapidly after the first treatment and remained near normal thereafter. Subsequent treatments were given because there remained roentgeno-

Fig. 3, Case 5
P. G. Acute infection lower lip. Before and after treatment.

Fig. 4. Case 7
G. McK. Furuncle of cheek. Result eight days after first treatment.

Fig. 5. Case 9
Wm. D. Erysipelas. Condition on admission and same patient twelve days later.

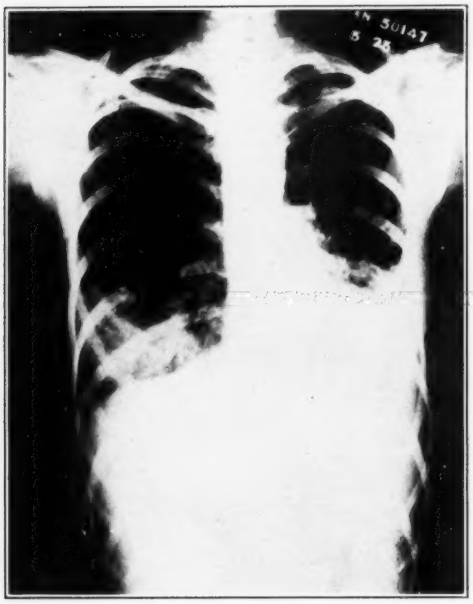

Fig. 6. Case 11
R. S. Infectious process in lung. Condition of the chest on day of first treatment.

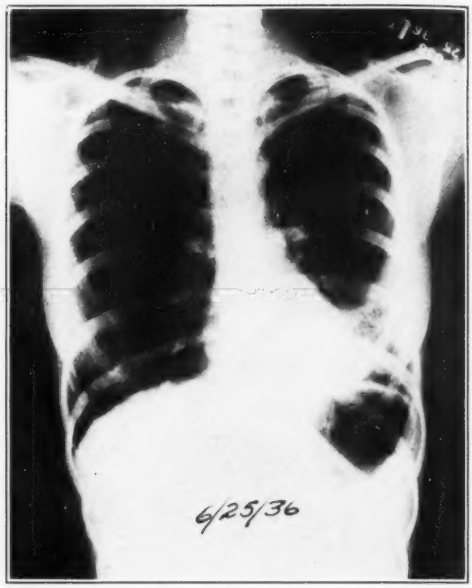

Fig. 7. Case 11
R. S. Infectious process in lung. Condition of the chest one month later. Organization has probably occurred in a portion of the left lower lobe.

graphic evidence of exudate. A certain amount of tissue change has remained in the left lower lobe, but the right side has cleared completely. From sputum cultures it was found that *Streptococcus viridans* was the predominating organism.

SUMMARY

The roentgen treatment of infections has not received the attention it deserves in this country. It has a wide field of usefulness as an adjunct to surgery and other procedures. The best results are obtained when the lesions are treated early, because in this stage many of the suppurative types are aborted. Definite benefit comes from its use in any stage.

The results are prompt, pain-relieving and effective in shortening the course of the disease.

The rules governing the treatment are clear and concise and vary only slightly, regardless of the lesion, its stage of development, or location. It is limited to the area of involvement and only small doses, from 10 to 20 per cent of an erythema dose, should be given. The quality of the ray may be varied for superficial and deep lesions, but the quantity only exceptionally.

This procedure does not interfere in any way with other methods or aids, except that the use of poultices, strong heat, or excessive cold is contraindicated.

The action of the rays in these conditions is not fully understood, but there is much experimental evidence that nature's defense is greatly augmented. The clinical data, however, are more convincing and important.

The papers mentioned in the bibliography support the statements in this communication and will furnish the reader a more extended reference to the literature on the subject.

A number of cases are submitted with illustrations showing the reaction of different types of infection to irradiation.

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THE BONE CHANGES IN SICKLE CELL ANEMIA*

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In this paper an attempt will be made to summarize the known facts concerning the pathology and roentgenographic appearance of bones in sickle cell anemia.

The material on which the work is based consists of the recorded observations of other investigators[1-37] and observations made at the John Gaston Hospital in Memphis. Thirty-nine Negroes with active and severe sickle cell anemia have been studied clinically. Eight autopsies have been performed. In each necropsy the bone marrow was examined, in five the skull was examined, and in three necropsies one or more long bones were sectioned. The pathologic material has been supplemented by microscopic sections of bones kindly furnished by Drs. E. C. Smith, W. R. Matthews, B. Sternberg, J. C. Corrigan, C. Brown and H. S. Thatcher. The bones of 30 Negroes with sickle cell anemia have been studied roentgenologically, the total number of roentgenograms being 259. For controls the files of the fracture clinic were used as these furnished a readily available source of bone pictures of healthy Negroes.

THE BONE MARROW

Numerous examinations of the bone marrow have been made in sickle cell anemia.[1 16] There is general agreement that the marrow is hyperplastic and that the flat and cancellous bones of the trunk and the shafts of the tibia and femur are filled with red marrow (*Fig. 1*). In the available literature no mention is made of dissection of the bones of the arm, wrist, hand and foot. In one of our necropsies on a 29-year-old Negro, hyperplastic marrow was found in the bones of the calvarium, scapula, clavicles, sternum, ribs, ilium, humerus, radius, ulna, tibia and fibula. In this cadaver patchy areas of red marrow were found in the larger cancellous bones of the foot, but no red marrow was found in the smaller, more distal bones of the foot nor in the bones of the wrist nor in the long bones of the hand.

The color of the marrow varied, depending on the degree of congestion, the amount of fat and the pathologic changes that were present, but the usual color was a uniform dark red to purplish black. The marrow from the shafts of most of the long bones had the consistency of soft jelly and was readily scooped out of the medullary cavity. In some of the long bones the trabeculae were numerous in the shaft, making marrow removal difficult and decalcification necessary before sections could be made for microscopic examination.

Microscopically the bone marrow presented a picture of great cellularity and congestion. The capillaries and the tissue spaces were filled with greatly elongated, pointed and curved (sickled) erythrocytes and in some sections the scattered islands of regenerating cells appeared as tiny islands in a sea of blood. The red color of the marrow is in part explained by this blood stasis and is not entirely due, as is often assumed, to excessive erythrogenesis. The predominant nucleated cells were of a primitive, undifferentiated type. Neutrophilic and eosinophilic granulocytes in various stages of maturation were conspicuous. Islands of megaloblasts and nucleated red blood cells were scattered throughout. Most of the nucleated erythrocytes were round, but sickled nucleated erythrocytes were demonstrable. Megakaryocytes were present in increased numbers. There were occasional lymphocytes

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*This study was originally started by Drs. Pulliam and Diggs in 1931. Dr. Pulliam died in 1933. Since that time the study has been carried on with the assistance of Dr. King, Radiologist of the John Gaston Hospital.

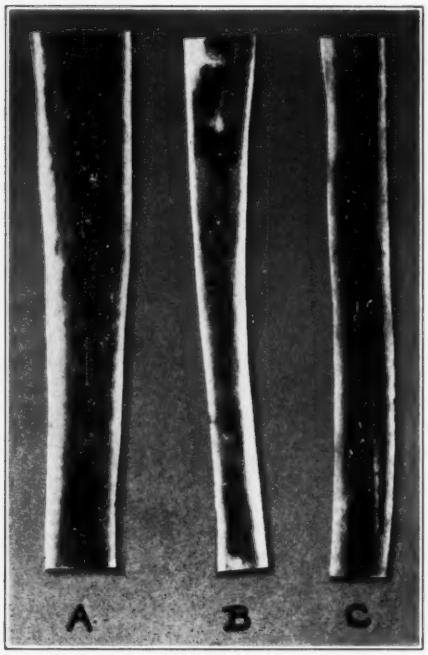

Fig. 1
Sickle cell anemia. Longitudinal sections of: (A) tibia, (B) tibia, (C) humerus. Hyperplastic marrow. Irregularities in thickness of cortex. New bone formation within medullary cavity.

and cells of the large mononuclear type, the latter sometimes containing within their cytoplasm ingested red blood cells, nuclear fragments, and brown and black pigment granules giving a variable iron reaction. Golden yellow and brown granules were found in small quantities in the lining cells of the capillaries. Special stains revealed an increase in reticulum. Usually there was no fat in the sections from sternum, ribs or vertebrae, but the marrow from the long bones contained variable amounts of fat.

In addition to the abnormal hematopoiesis and blood stasis there has been recorded in the literature and repeatedly confirmed in our material evidence of degenerative and reparative processes in the marrow. Lesions found included thrombosis, infarction, necrosis, hemorrhage, granular and crystalline pigment deposits, hyalinization, fibrosis, abnormal calcification, and new bone formation. These lesions in the bone marrow are similar to those found in other organs of the body, the essential features of which are congestion, thrombosis and connective tissue replacement of parenchyma.

THE BONE STRUCTURE

In contrast to extensive studies of the bone marrow in sickle cell anemia there have been few studies of the structure of the bones. The earliest and most important contribution on the subject was by Graham,[1] who sectioned the long bones of the leg in a 30-year-old Negro with sickle cell anemia and noted gross distortion of the anatomic pattern, scarring and new bone formation within the marrow cavity. In the light of present knowledge, the bone changes described by Graham represent the end stage in a progressive osteosclerotic process. Similar but less striking changes have been reported by Mathews.[16]

In our necropsy material the long bones appeared to be of normal size. The surfaces of the bones as a rule appeared normal, but gross roughening and irregular periosteal new bone formation was observed in some of the bones, being most marked in the tibia and fibula of the older patients with leg ulcers. The density of the cortex varied considerably in different bones and in different parts of the same bone. In general, the shafts of the long bones were well calcified, the cortex thicker than normal and sectioning difficult. On section the hyperplastic marrow was the most conspicuous feature. The red marrow was not confined to the medullary cavity, but filled the widened haversian canals of the thickened cortex, giving it a honeycombed appearance. The marrow invasion of the cortex was most marked at the inner margin, but in some cases extended to the periosteum. New bone formation within the shafts of the long bones was the rule, usually taking the form of delicate linear trabeculae, but in some instances taking the form of concentric layers of lamellated bone adjacent to the cortex and parallel to it or of thin sheets of bone perpendicular to the cortex. In occasional sections there was complete replacement of the marrow cavity (*Fig. 2*).

The cancellous bones of the trunk and the parietal and frontal bones of the skull as a rule were sectioned with unusual ease, indicating defective calcification. The skull was described as of normal thickness in one case and in four others the maximal thickness of the parietal bones was 4, 4, 12 and 20 mm., respectively. The inner and outer tables were less distinct than normal; the cancellous spaces were large and filled with red marrow. No perpendicular arrangement of trabeculae was noted. The inner

and outer surfaces of several skulls were purplish in color, probably due to the large amount of blood within the cancellous spaces and to thinness of the inner and outer tables. The sternum and ribs appeared to be of normal size, and on section presented no gross abnormality.

ROENTGENOGRAPHIC APPEARANCE

Most of the information concerning the bone changes in sickle cell anemia has been obtained from roentgenographic studies.[6, 8, 12, 13, 15 and 17-37] Abnormal bone changes as revealed by x-ray were first observed by Grullee and Rose and reported by Cooley, Witmer and Lee in 1927.[17] Since that time numerous observations have been placed on record. The literature appearing in the radiology journals is somewhat misleading, for the unusual and striking findings are emphasized and the negative findings ignored. Also there is a tendency to overstate the possibilities of x-ray in differential diagnosis. An analysis of the x-ray findings in the case reports of sickle cell anemia reveals a more representative picture. The most important articles dealing with the roentgenographic changes in bones are by Rose,[18] Grinnan,[21] Moore,[25] Leivy and Schnabel[26] and Brandau.[32]

The roentgenograms of bones of the majority of patients with sickle cell anemia reveal no abnormality in size, shape or density. The roentgenograms of some patients show moderate changes and a few show striking lesions. The bones most likely to reveal abnormality are the skull, vertebrae and tibia and fibula (*Table 1*).

Skull.—The skull changes usually described and considered characteristic are: increase in thickness, absence of well defined outer table, osteoporosis and pointed trabecular striations

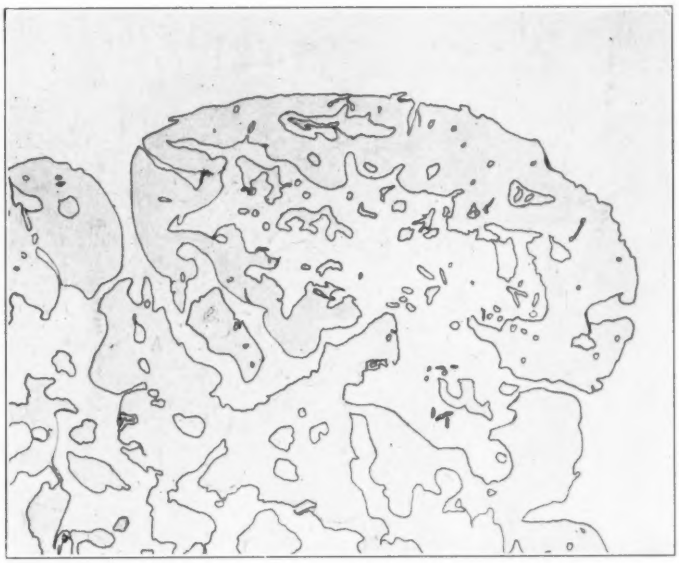

Fig. 2

Sickle cell anemia. Drawing showing microscopic appearance of shaft of fibula. Marrow replacement by osteoid tissue and new bone. Irregularities in margin and in thickness of cortex (reduced from magnification X 15).

Fig. 3

Sickle cell anemia. Skull of 19-year-old Negro. Osteoporosis of vertex, diploic widening, absence of distinct outer table, "hair-on-end" trabeculations.

radiating outward from the inner table and perpendicular to it (*Fig. 3*). The thickening of the diploe is bilaterally symmetrical, usually is limited to the parietal bone, but may also involve the frontal and occipital bones. The thickness is maximal at the vertex and tapers off to normal as the temporal region is approached. This type of skull abnormality is similar to that described in erythroblastic anemia, in hemolytic jaundice, in the skulls of ancient Myan Indians and in other conditions. Hence it is not specific for sickle cell anemia. Only 9 instances of this type of abnormality in sickle cell anemia patients have been described in the literature, whereas its absence has been noted in 16 cases. In our series of 30 cases, thickening of the skull with perpendicular striations was striking in one (*Fig. 3*), and demonstrable but not marked in 3 others. The age range for all patients with this type of abnormality is from 5 to 25 years, the most marked type of abnormality being found in the older children and adults. The average age of patients with this lesion is 14 years. This type of abnormality is one of osteoporosis with widening of the marrow spaces, decalcification and thinning of the cortex, and is not necessarily associated with significant osteosclerotic changes in the long bones.

Other changes described in the skulls of patients with sickle cell anemia and observed in our series are thickening of the diploe with ill defined tables, or osteoporosis with indistinct striations between the tables. In many cases the vertex has a granular appearance (osteoporosis), and in occasional cases there are roundish areas of increased radiability, varying in size and in

Table 1

DEGREE OF BONE PATHOLOGY AS REVEALED IN ROENTGENOGRAMS. (— NO ABNORMALITY, ? QUESTIONABLY SIGNIFICANT, + DEFINITE, ++ STRIKING)

Name	Age	Hbg. Gm.	Leg Ulcer	Tibia Fibula	Femur	Humerus	Radius Ulna	Hands Feet	Skull
J. W.	1	6.9	—	—	—	—	—	—	—
B. H.	2	7.1	—	—	—	—	—		?
M. S.	3	8.0	—	—	—				—
M. S.	3	7.4	—	—	—				—
C. T.	6	7.4	—	—	—	—			—
K. M.	7	7.4	—				—	—	—
M. B.	9	6.3	—	++	?	—	—	—	—
W. M.	9	6.8	—	—	—	—	—	—	++
D. G.	9	7.7	—	?	—	—		—	—
P. M.	12	6.8	+	?	—	—		—	—
M. F.	12	8.5	+	++	—				?
W. O.	13	9.0	—	—		—	+	—	?
R. C.	14	8.6	—	+	—	—	—	?	—
A. M. N.	15	7.2	—						—
J. K.	16	8.4	—	—					—
L. Q. B.	16	7.5	—	?			—	—	—
S. A.	16	9.4	—	?		—		—	—
M. B.	18	7.6	—	?		—			—
L. C.	18	7.0	+	++	—	—		—	—
J. R.	18	6.5	+	+++				—	+
A. M. M.	18	6.5	—	+	—	?	—		—
L. M.	19	5.2	+	+	—	?	?	—	+++
T. R.	19	8.0	—	?			+	+	+
B. W. J.	20	7.9	—	?					—
W. T.	21	7.5	—						—
L. J.	23	7.8	+	?			—		—
J. W.	26	3.6	++	+++			++		?
C. S.	29	3.6	++	+++	+	++	++	—	?
L. B.	31	8.0	—	+		—	—	—	—
G. C.	51	3.7	++	++++					+

distribution and resembling somewhat metastatic malignancy or multiple myeloma (*Fig. 4*). Measurements of the mid-frontal and mid-parietal thickness of the skull in normal Negroes and in sickle cell anemia patients reveal wide variations in both groups and no consistent differences between the two groups. The roentgenographic appearance of the bones of the face is normal.

Bones of Trunk.—The scapula, sternum, clavicles, ribs and pelvic bones present no unusual or diagnostic features. Moderate osteoporosis is the rule. There is no appreciable widening of the bones or deformity. Osteosclerotic changes are seldom seen.

The vertebrae in sickle cell anemia show as do the other cancellous bones diffuse and localized variations in radiability. In addition there is, in adults with the disease, flattening of the vertebral bodies[26] (*Fig. 5*). Average measurements of the height and width of the first four lumbar vertebral bodies in the anterior-posterior and lateral views revealed a greater height-to-width ratio in sickle cell anemia patients than in normal controls (*Table 2*). The compression of vertebrae is explained on the basis of weight bearing on poorly calcified bones.

Long Bones.—Moderate widening of the medullary cavity and of the diameter of the shaft, expansion of the diaphyseal extremities, increased radiability, prominent trabeculations and increased density of the zone of ossification

Fig. 4

Sickle cell anemia. Skull of 51-year-old Negro. Irregularities in density of calvarium, with localized areas of decreased density.

Table 2

RATIO OF HEIGHT TO WIDTH OF VERTEBRAL BODIES AS DETERMINED FROM ROENTGENOGRAMS OF SICKLE CELL ANEMIA PATIENTS AND OF NORMAL NEGROES OF COMPARABLE AGE.

ANTERIOR-POSTERIOR VIEW			
Sickle Cell Anemia			Controls
Patient	Age	Ratio	
R. C.	14	1 to 2.2	
M. B.	18	1 to 1.6	
A. M. N.	18	1 to 1.8	In 14 normal unselected Negro adults the ratio of height to width was on the average: 1 to 1.4.
L. M.	19	1 to 1.8	
W. T.	21	1 to 1.8	
L. B.	31	1 to 1.4	
G. C.	51	1 to 2.8	
LATERAL VIEW			
K. M.	7	1 to 1.1	
W. O.	13	1 to 1.2	
J. C.	16	1 to 1.7	
L. Q. B.	16	1 to 1.3	In 14 normal unselected Negro adults the ratio of height to width of vertebral bodies was on the average: 1 to 1.2.
B. J.	20	1 to 1.6	
L. J.	23	1 to 1.4	
J. W.	26	1 to 1.5	
C. S.	29	1 to 1.8	

have been described in the long bones of children with sickle cell anemia, but marked increase in width, notable thinning of the cortex or generalized porosity which are characteristic of the long bones in erythroblastic anemia have not been observed. In our series the roentgenograms of the long bones in infants and of young children reveal no significant changes. In older children and in adults with the disease roentgenograms are likewise normal in appearance as a rule. When abnormalities are present they take the form of osteosclerosis, with cortical thickening,[25] new bone formation within the medullary cavity and patchy irregularities in density and pattern of the bone structure without an increase in the bone diameter.

Fig. 5
Sickle cell anemia. Moderate flattening of lumbar spines.

Mid-shaft measurements were made of the roentgen shadows of the tibia, fibula, radius, ulna and first phalanx of the index finger of patients with sickle cell anemia and of normal Negroes of comparable age. The technic used in making the radiographs was the same in both series. The total number of bones measured in the sickle cell anemia series was 82 and in the control series 237. Variations were found in the measurements of the individual bones in both series with a wider range of variation in the sickle cell anemia group. The average measurements of all bones studied and in all age groups disclosed no significant difference in the diameters of the long bones in the two series. There was a tendency for the long bones in sickle cell anemia to be more narrow than normal. The percentage of bone diameter occupied by the medullary cavity tended to be slightly greater in the sickle cell anemia group, but the actual width of the shadow cast by the marrow canal was in the two groups essentially the same. The measurements for adults are summarized in *Table 3*. The small number of patients examined in the various age groups, the inaccuracies involved in the estimation of the size of bones by their roentgenographic appearance and the limitations of average figures in expressing the true picture prevent the taking of these measures too seriously, but at least they lend no support to the oft-repeated statement that the medullary cavity and bone diameter are characteristically widened in sickle cell anemia.

No measurements were made of the length of the long bones in comparison with normal, but

Table 3

AVERAGE MID-SHAFT DIAMETER OF LONG BONES AND PERCENTAGE OF DIAMETER OCCUPIED BY MEDULLARY CANAL DETERMINED BY MEASUREMENTS OF ROENTGENOGRAMS OF ADULTS WITH SICKLE CELL ANEMIA AND ROENTGENOGRAMS OF NORMAL NEGROES

Bone	Number of Cases		Average Diameter Millimeters		Average Percentage of Diameter Occupied by Medullary Cavity	
	Normal	Sickle Cell	Normal	Sickle Cell	Normal	Sickle Cell
Tibia	30	14	25	25	45	56
Fibula	30	14	18	14	43	49
Radius	31	6	14	14	41	50
Ulna	11	6	13	12	44	43
Proximal phalanx—index finger	19	6	11	9	52	56

Fig. 6
Sickle cell anemia. Beginning osteosclerotic changes in tibia and fibula.

the length appeared to be increased. It is a well established fact that older children and adults with sickle cell anemia are, as a class, tall and slender and that they have long arms, legs, fingers and toes.

The bones of the extremities most likely to show abnormalities are the tibia and fibula, but even in these bones there is often no demonstrable change. In the literature negative roentgenographic changes in the lower extremity are recorded in 18 instances and positive findings in 9. In our series of 27 cases, there were no changes in 8, questionable changes in 8 and definite changes in 11. The changes were most marked in adults with the disease and were least likely to be positive in children (*Table 1*). In the reported cases the average age of those showing no roentgenographic changes is 8 years, and the average age of those with positive findings is 23 years.

The sclerosing lesions in the tibia and fibula present a variable roentgenographic picture. The most common abnormalities are thickening of the cortex, narrowing of the medullary cavity and abnormal trabecular markings (*Fig. 6*). The cortex may be diffusely and fairly uniformly thickened, but in some cases the thickening is limited to one area, usually the middle of the lower half. In other cases the thickness of the cortex varies in different parts of the same bone (*Figs. 7, 8* and *9*). Often longitudinal lines of decreased density are observed in the thickened cortex, giving to it a spongy appearance (*Figs. 7* and *8*). In some bones the cortex and the medullary canal seem to fuse, giving the picture of a cancellous shaft with coarse, linear markings (*Figs. 8* and *9*).

The trabecular markings at the ends of the tibia and fibula are less uniform in width and density than normal and the pattern is less orderly. Multiple and parallel transverse lines of increased density are commonly seen at the lower end of the tibia. The predominant direction taken by the trabeculations in the shaft are par-

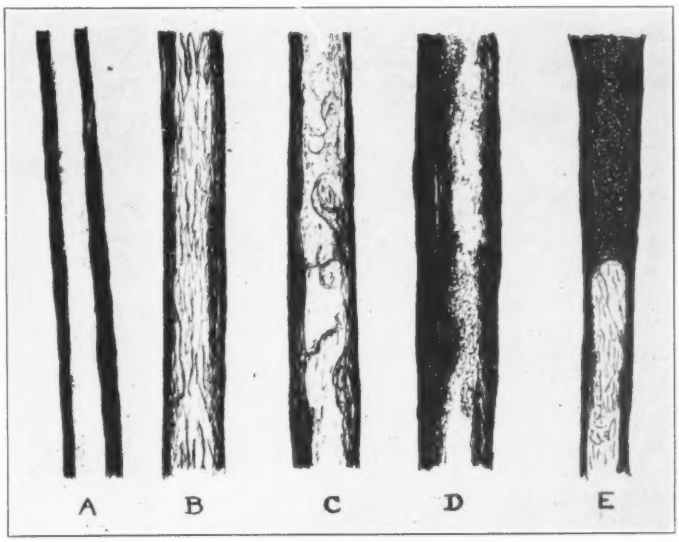

Fig. 7
Sickle cell anemia. Tracing of roentgenograms of fibulae showing: (A) no change, (B) osteoporosis with increase in trabecular markings, (C) irregularities in thickness of cortex. Abnormal bone formation within medullary cavity, (D) cortical thickening. narrowing of medullary cavity, (E) complete obliteration of marrow cavity (the microscopic appearance of this lesion is given in Fig. 2).

Fig. 8

Sickle cell anemia. Right and left tibia in 26-year-old Negro with chronic leg ulcers. Widening and bowing. Indistinct boundaries between cortex and medullary canal of tibia. Irregularities in thickness of cortex of fibula. Abnormal trabecular markings. Periosteal roughening.

allel to the longitudinal axis, but perpendicular and slanting striations and spoke-like and curved lines radiating out from a common cortical center are observed (*Fig. 7*).

In the end stages of the sclerosing process the bone structure is grossly disarranged and the normal relationship of medullary cavity and cortex is lost (*Fig. 9*). Bizarre combinations of increased radiability and density are found which simulate the changes found in chronic inflamations or cystic disease.

Chronic leg ulcers are common in adults with sickle cell anemia. In association with long-standing inflammation of the skin, edema and limitation of movement the diameter of the tibia and fibula is wider and osteoporosis is likely to be more marked (*Fig. 8*) than in patients of the same age without ulcer or in the uninvolved leg of the same patient. Thus it is seen that the roentgenographic findings in the tibia and fibula in association with leg ulceration present an exception to the general rule.

The changes found in the femur, humerus, radius and ulna are similar to those found in the tibia and fibula, the essential features of which are abnormal trabeculations, osteosclerosis, narrowing of the medullary cavity and patchy irregularities in bone density. Usually the osteosclerotic changes are more marked in the tibia and fibula than in the other long bones, but there are exceptions.[25]

The bones of the wrist, hands, ankles and feet present no striking roentgenographic changes. Osteoporosis of a moderate degree has been noted in the cancellous bones of the hands and feet, but this is not a constant feature. In older patients there is a thickening of the compact cortex of the long bones of the hands and feet.

Usually in sickle cell anemia there is no periosteal involvement, but we have observed in the roentgenograms of three children with sickle cell anemia elevation of the periosteum in one or more long bones. Irregularities in the outer margin of the cortex and periosteal new bone formation are observed in the older patients with advanced osteosclerotic lesions (*Fig. 9*). Path-

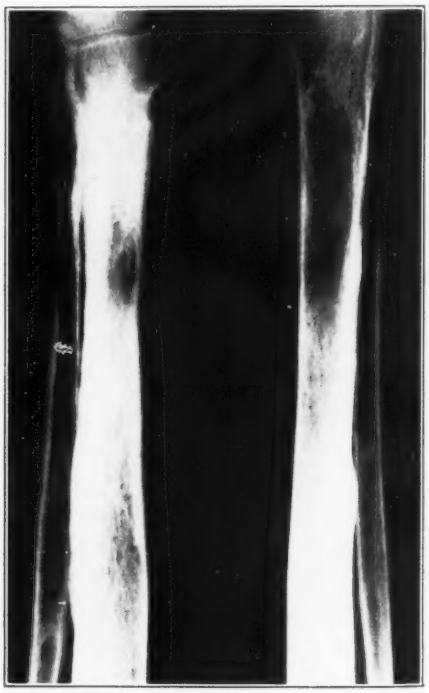

Fig. 9

Sickle cell anemia. End stage osteosclerosis in 51-year-old Negro. Patchy areas of increased radiability. Gross distortion of anatomic pattern. Periosteal new bone formation.

ologic fractures have not been reported in the literature or observed in our series. In a patient with bilateral leg ulcers of long standing there was moderate bowing of the tibia and fibula (*Fig. 8*). It is probable that future studies will reveal pathological fractures and deformities in the long bones in occasional cases.

No significant abnormality in the appearance of ossification centers was noted in the small number of children available for study. In older children and young adults about half revealed evidence of a significant delay in the closure of epiphyses.

Miscellaneous.—The degree of bone change appears to have no relationship to the severity of the anemia (*Table 1*). The osteosclerotic type of bone abnormality is seldom seen in young children, begins to appear in later years of childhood and is maximal in adults. Since sickle cell anemia usually becomes manifest in the early years of life and continues thereafter, it follows that the osteosclerotic lesions are correlated with the duration of the anemia. The majority of patients with sickle cell anemia die in the first two decades and do not live long enough to develop osteosclerotic changes in their most characteristic forms. It is difficult to correlate the osteoporotic, bone-widening and cortex-narrowing processes with the age of the patients or severity of anemia, but these changes seem to be more marked in the younger patients and are less in evidence in adults. Re-examination of the bones of the same patients over extended periods of time are indicated in order to work out the time and sequence of appearance of lesions and to follow the progress of lesions.

The bones of Negroes with the sickle cell trait in absence of the specific hemolytic anemia (sicklemia) are negative on roentgenologic examination. Since the x-ray findings may also be negative in patients with sickle cell anemia, it follows that roentgenographic studies are of little value in the differential diagnosis of these two conditions.

It is our opinion that the bone changes in sickle cell anemia are, even in their most marked form, non-specific. At least we have no confidence in our ability to diagnose the condition from roentgenograms alone. Thousands of x-rays of the bones of Negroes are made each month at the John Gaston Hospital and often lesions which we thought characteristic of sickle cell anemia were noted, but on examining the clinical record or making further laboratory studies we were able to confirm the roentgen diagnosis in only one case. This diagnosis was made by Dr. Pulliam on the basis of abnormal trabeculations in the tibia of a Negro with leg ulcer. In spite of the limitations of roentgenograms in diagnosis and in confirmation of diagnosis, it is essential that studies be continued in order to advance knowledge and to familiarize radiologists and clinicians with the possibilities of the disease. The use of x-rays as a substitute for other more reliable and less expensive diagnostic measures, or routine roentgenograms on suspected cases is unjustified.

Patients with sickle cell anemia often have attacks of severe and migratory joint pains with associated fever and leukocytosis leading to the diagnosis of acute rheumatic fever or infectious arthritis. The symptoms subside in a few days or weeks, leaving no residual deformity or demonstrable x-ray changes. Patients with sickle cell anemia also are subject to attacks of severe pains in the bones, often with localized bone tenderness which, together with the characteristic fever and leukocytosis, leads to the diagnosis of osteomyelitis and to emergency surgery. On opening the bone there is no exudate and the cultures are sterile. It is thought that these symptoms and signs of inflammatory reaction in bones and joints are due to circulatory abnormalities such as severe stasis, multiple capillary thrombi and in some instances to thrombosis of larger vessels with infarction and necrosis.

Although the diagnosis of acute infections of the bones and joints is often erroneous, this does not prevent patients with sickle cell anemia from infections of the osseous system. On the contrary, bone and joint infections are more common than they are in normal individuals. In our series of 30 patients roentgenologically studied, osteomyelitis due to staphylococcus infection was present in one and suppurative arthritis due to paratyphoid B in another. In three additional patients there was limitation of movement and x-ray evidence of chronic joint involvement, the joints affected being the shoulder in one case and the hip in two cases.

Cerebral thrombosis and degenerative spinal cord changes are found in a few patients with sickle cell anemia, and in association with these central nervous system lesions there is muscle and bone atrophy.

SUMMARY

The gross, microscopic and radiologic appearance of bones in sickle cell anemia indicates that the bone marrow is primarily involved. Two factors appear to be working in opposite directions, one the hyperplastic marrow tending to increase its volume at the expense of bone and the other the sclerosing factors tending to replace the marrow and to substitute in its place osteoid tissue or new bone. The excessive and abnormal growth activity of the marrow has as a rule no significant effect upon the bony cage within which it is confined, and such effects as it does have are maximal in the bones which are normally engaged in blood cell formation such as the skull and cancellous bones of the trunk. In these bones osteoporosis is dominant. In the long bones where normal hematopoiesis does not occur, sclerosis is dominant. In the most distal bones of the hands and feet where marrow is absent the changes are minimal. The combination of marrow expanding and marrow replacing processes leads, if the patient lives long enough, to gross anatomical derangements in the bone structure.

The photographs were made by Dr. Joseph L. Scianni, Artist, University of Tennessee, Pathological Institute.

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DISCUSSION (*Abstract*)

Dr. Maxwell M. Wintrobe, Baltimore, Md.—Dr. Diggs has presented a very able and thorough report. His observations raise several questions of interest. One of these is concerned with congenital hemolytic jaundice. This is a disease which in many respects resembles sickle cell anemia. Both disorders seem to be due to a congenital structural abnormality of the red corpuscles, which is inherited possibly as a simple mendelian dominant. In both conditions there is marked hyperplasia of the bone marrow. If hyperplasia is the cause of the bone changes found in sickle cell anemia, it would be of interest to know whether similar findings are as often present in hemolytic jaundice.

Dr. Diggs has mentioned the pains and arthritic manifestations commonly seen in patients suffering from sickle cell anemia. Sir William Osler called syphilis "the great imitator." In the same sense sickle cell anemia may be called "the little imitator" for the symptoms of this disease have often led to a mistaken diagnosis. Symptoms of abdominal pain and prostration have often led to the diagnosis of an "abdominal emergency." Again, pain when about the joints has led to the erro-

neous diagnosis of rheumatic fever. Such a diagnosis is often all the more convincing because these patients may present marked cardiac signs without demonstrable lesions at autopsy. We have been particularly impressed by cases in which competent cardiologists have made the diagnosis of mitral stenosis and this diagnosis has been supported by cardioroentgenograms as well as by electrocardiogram. In six of the cases at the Johns Hopkins Hospital, first degree heart block has been found. Yet no evidence of rheumatic heart disease, and for that matter, no explanation of the cardiac signs has been found at autopsy.

Dr. V. P. Sydenstricker, Augusta, Ga.—Dr. Diggs has presented the first comprehensive study of the bone changes in sickle cell anemia and has correlated the gross and microscopic findings with the roentgenological appearance. Until now both the gross and histologic pathology of the bones has been neglected, changes in the hemopoietic elements of the bone marrow have had much attention but no one has gone into the nature of the disturbances in ossification which cause alterations in the x-ray picture. The essayist's description of chronic congestion with thrombosis, fibrosis and eventual sclerosis is excellent and presents a process analogous to that which occurs in the spleen. His demonstration of hyperplasia of all the marrow elements with invasion of the normal architecture of the bones and later imperfect repair, gives the complete explanation of the marked variations found on x-ray examination. My own belief is that there is rather direct relation between the age of the patient, the severity and duration of anemia and the degree of osseous damage. In older patients there has been much more opportunity for repair. Cranial osteoporosis is apt to persist long after long bone changes have been restored. In our clinic the x-ray report "osteoporosis of the skull suggestive of sickle cell anemia" has in several instances led to the demonstration of sicklemia in adult patients with no symptoms referable to the disease. We have felt that such individuals are "recovered cases" of sickle cell anemia. As Dr. Diggs and Dr. Pierson have pointed out, the radiographic findings are non-specific but warrant careful survey of the blood. Not infrequently young children with severe anemia show little or nothing in their bones, we think because the process of marrow hyperplasia has not persisted long enough.

The possible importance of the radiographic bone changes to ethnologists and paleopathologists is considerable. Moore first noted the frequent finding of osteoporosis in old Peruvian and Mayan skulls and called attention to its similarity to the finding in sickle cell anemia. Apparently no similar lesion has been found in North American Indian skulls. It is of great interest that recent reports would indicate that sickle cell anemia and sicklemia are not rare in Mexican Indians of possible Mayan ancestry and also that sicklemia was not found by McGiboney in the examination of a thousand North American Indians. The idea that sickle cell anemia is limited to the Negro race seems no longer tenable. Certainly it is much more frequent among Negroes and in the isolated instances reported among white people it is perhaps impossible to rule out some remote mixture of blood. It seems unlikely that there could be any wide distribution of Negro blood among Mexican Indians.

Dr. Diggs is to be congratulated on his excellent clarification of an important phase of sickle cell anemia.

Dr. Diggs (closing).—In erythroblastic (Cooley's) anemia there is an increase in the width of the medullary canal and diameter of all the bones of the body, thinning of the cortex, increased radiability and prominent linear markings. The osteoporosis and widening of the bones of the distal extremities and the perpendicular striations of the skull are in this disease striking. Erythroblastic anemia usually becomes manifest in infancy or early childhood and follows a progressive down-hill course. Rarely do those afflicted attain puberty. The characteristic alterations in the bone structure develop early, become more marked as time goes on and are demonstrable in practically all of the cases.

Few reports are available concerning bone studies in hemolytic jaundice. Changes comparable to those found in erythroblastic anemia have been noted in a few cases, but in some of these cases the accuracy of the diagnosis is open to question. The majority of patients with congenital hemolytic jaundice have no demonstrable alterations in their bones or the changes are minor in degree. Tower skull, infantilism and congenital defects of various sorts have been described in patients with this disease. Because of the inaccuracy of diagnoses, the scarcity of reports and the lack of uniformity of the findings, we are as yet unable to say with assurance what the characteristic bone lesions in hemolytic jaundice are.

Erythroblastic anemia, hemolytic jaundice and sickle cell anemia have in common an inherited bone marrow defect, anemia which appears early in life, marrow hyperplasia and a shift in the cellular constituents of the blood toward primitive and less differentiated cell types. These three diseases, despite their similarities, are different in many respects. Sickle cell anemia occurs predominantly in Negroes. The erythrocytes are elongated, angular and multipointed. There is in sickle cell anemia a tendency toward capillary stasis and thrombosis which is not present in the other congenital anemias. Under conditions of anoxemia the cellular distortion is exaggerated and in tissues such as the bone marrow where the oxygen tension is normally low the abnormality in shape is maximal. It is thought that the shape of the cells together with the increase in white blood cells and in platelets interfere with the circulation of blood in the capillaries and favor intravascular coagulation. The sclerosing and patchy lesions found in the bones of patients with sickle cell anemia and the painful manifestations of the disease are explainable on the basis of sluggish blood flow, capillary thrombi and in some cases thrombosis of the larger vessels with infarction and necrosis. These changes have been found in the brain, lung, spleen and kidney of patients with sickle cell anemia and it is logical that they should likewise be demonstrable in the bone marrow. The cortical thickening of the long bones of the extremities and the new bone formation within the marrow cavity are explainable on the basis of vascular stasis.

THYROIDECTOMY*

FACTORS INFLUENCING THE MORTALITY

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The aim of any operative procedure is to cure a diseased condition without endangering the patient's life. This is an ideal rarely to be attained, but constantly to be sought. Professor Halsted,[1] in "The Operative History of Goitre," tells of the years of struggle to rid man of an "unsightly and frequently fatal disease" and one "accepted as an inoperable affliction." Although thyroidectomy can now be performed with relative safety by many surgeons, the mortality ranges between 2 and 5 per cent. It is with this small percentage that our interest lies, and it is the object of this paper to discuss this mortality.

Death following thyroidectomy is usually due to one of three causes: first, conditions due to the disease itself, as postoperative hyperthyroidism with or without cardiac failure; second, technical errors, as hemorrhage or trauma; and third, to conditions which may follow any operation, such as pneumonia, embolism, or infection.

Postoperative hyperthyroidism is dependent on the degree of thyroid toxicity present before operation, and cardiac failure is the result of long-continued stimulation and overactivity of the heart. Most patients dying after operation have suffered from hyperthyroidism for several years and have usually been treated with iodine for a long period of time. They are usually over 40, and the goiter is more often nodular (the so-called toxic adenoma) rather than the diffuse hyperplastic type (exophthalmic goiter). There is usually a history of loss of weight, with nervousness, hysteria and palpitation. Nausea and diarrhea are not uncommon. On examination they are nervous and hysterical, the pulse is rapid and frequently irregular, the skin is moist and soft, and the basal metabolic rate greatly increased.

In addition, Lahey[2] has described under the name "apathetic hyperthyroidism" a type which presents a particularly bad risk. These patients are past middle age and have had the disease for several years. Except for weight loss, the signs are noticeably absent. In fact, an entirely different state may exist with cool skin and a normal or slightly elevated pulse and metabolic rate. These patients have probably passed through many periods of thyroid overactivity, and Lahey believes that because of their age they are unable "to react by activation to excessive thyroid secretion."

Mortality following thyroidectomy can best be lowered by preventing preoperative hyperthyroidism. It is well known and generally accepted that a young patient who has suffered from hyperthyroidism for only a few months can be prepared for operation by the administration of Lugol's solution. Marine[3,4] and his coworkers showed as early as 1908 that the administration of iodine caused a hyperplastic gland to return to a colloid or resting state, and this has been confirmed by many others, notably Plummer,[5] and by Thomas and Rienhoff.[6] Along with the changes in structure of the gland, there is a remission of symptoms and signs and a fall in the basal metabolic rate. Young patients, in whom the disease has been present only a short period of time, and in whom a temporary remission has been effected by iodine, can be operated upon with comparative safety. It has frequently been pointed out that iodine should be used for preoperative preparation and for this purpose alone.

Fig. 1
Diffuse goiter with hyperthyroidism in a patient aged 51, present for five years. Marked loss of weight. Operation done in stages.

*Read in Section on Surgery, Southern Medical Association, Thirtieth Annual Meeting, Baltimore, Maryland, November 17-20, 1936.

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In spite of these statements and of repeated warnings, many physicians, either because of ignorance or prejudice, persist in using iodine for treatment. This is done with the idea of "trying medical treatment first," although what they intend to accomplish I have been unable to ascertain. Surely medicine has no place in the treatment of hyperthyroidism any more than it has in appendicitis. The indiscriminate and prolonged use of iodine brings about a state of chronic hyperthyroidism. In these patients the symptoms may be obscured, the further response to iodine, either before or after operation, is diminished, and the risk is immeasurably increased. Moreover, long hospitalization and the removal of the gland in stages imposes a severe economic burden. A case in point is as follows:

Mrs. F., a housewife of 51, entered the hospital in July, 1936, because of a goiter. Five years previously her condition had been diagnosed by her physician, who gave her iodine, which caused immediate improvement. A few weeks later her symptoms returned, but the use of iodine was continued. During the next five years her weight dropped from 150 to 84 pounds, and her nervousness and hysteria increased. Her eyes became markedly exophthalmic and her heart was rapid, forceful, and at times irregular. At the first examination I was unable to obtain a history because of her constant weeping. She was emaciated, the eyes were bulging, the pulse 150, and the basal metabolic rate +106. The thyroid gland was diffusely, but moderately, enlarged. The use of iodine was discontinued and she was given bed rest, sedatives, and a high caloric diet. In two weeks there was little change in her condition and Lugol's solution was again given. After three weeks her pulse dropped to 120, and her metabolic rate to +60. At this time both superior polar vessels were ligated. She stood this procedure well, but the pulse rose to 170 per minute during the operation. Within the next two weeks she gained 4 pounds and she was generally better. At the end of this time the right lobe of the thyroid gland was removed under cyclopropane anesthesia. Her postoperative course was stormy and the intravenous administration of iodine apparently had little effect. Her pulse rose to 160 and the temperature to 102°, but her symptoms gradually abated. In a month she was able to leave the hospital, somewhat improved, with a pulse of 110, and a gain in weight of 7 pounds. After a month of rest at home the left lobe was removed. This operation was followed by a less stormy course and at present, four months after entering the hospital, her weight is 107 pounds (a gain of 23 pounds), and her pulse ranges from 90 to 100 per minute.

This case is cited to emphasize the folly of medical treatment and the difficulties attendant upon treating such a patient.

Fig. 2

Recurrent hyperthyroidism five years after operation. Marked nervousness and loss of weight. Metabolic rate +60. Removal of thyroid in stages.

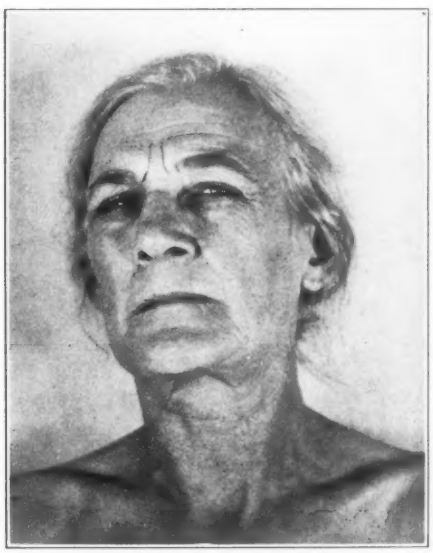

Fig. 3

"Apathetic" hyperthyroidism in a woman of 46 but appearing much older. Small nodular goiter with symptoms for a number of years. Metabolic rate +35. Removal in stages advisable in this type of patient.

Operation upon patients of this type should always be done in stages. Where iodine has been given over a period of years response to this drug is uncertain and the usual methods for controlling postoperative hyperthyroidism may fail. Therefore, preliminary ligation, both as a test of the degree of reaction and as well for its therapeutic effect, should be done. In a majority of instances patients improve following ligation, and in two or three weeks one lobe can be removed. Following removal of one lobe there is usually marked recession of symptoms and the final operation can be done in from two to six weeks. The type of operation, whether in stages or at one time, should be determined before the operation is begun, for patients who have taken iodine for a long period are apt to appear better than they really are, and suffer from a severe postoperative crisis. In short, stage operations should be considered in patients with marked toxicity or those who have had the disease for several years. This includes those whose age, or apparent age, is over 40, who are emaciated, or whose basal metabolic rate is unusually high.

Preparation for the operation should be carefully carried out. A high caloric diet, with carbohydrates predominating, should be given during the period of preparation and resumed as soon as possible after operation. A sedative, such as sodium iso-ethylamyl barbiturate, is given the night before operation and morphine and scopolamin on the morning of operation. I am convinced that a general anesthetic should be used rather than local infiltration. The mere knowledge that an operation is to be done will frequently cause the pulse to rise to 150 or more, and the nervous strain of the operation itself will elevate the metabolism to the point of causing a severe crisis. Cyclopropane, because of the amount of oxygen administered with it (70 to 80 per cent) will largely overcome the dangers of anoxemia which is frequently seen in these cases, and is the anesthetic of choice. Operation should be so carried out that it can be stopped at any point, and for that reason vessels should be tied when cut. A rise in the pulse rate which persists, a sustained rise in pulse pressure, difficulty of respiration, or unusual technical difficulties are indications for removal of the gland in stages, or even for its abandonment for a time.

Technical difficulties, such as hemorrhage, injury to the trachea or the recurrent laryngeal nerves, are more apt to occur in large colloid goiters. This type should be examined by roentgenogram before operation, and if the trachea is markedly compressed or deviated, the operation should be done in stages. Otherwise, the trachea rings, softened by long pressure, may collapse when the support of the gland is removed.

Thyroidectomy should be carried out with great care, since injury to the trachea and the recurrent laryngeal nerves, or excessive hemorrhage, may cause death. While these patients do not stand prolonged operations well, speed should not be sacrificed for care, and they undergo a long operation far better than they do excessive trauma or a postoperative hemorrhage.

The operation perfected by Halsted has not been improved upon. With it practically all the gland is removed except a small portion along the posterior capsule at the inferior poles. I believe that the use of fine silk for ligatures results in less inflammation and, therefore, in less postoperative reaction in the toxic patient. I am sure, from experience, that the use of silk and the closure of the wound without drainage reduces the incidence of infection and leads to an easier postoperative course.

Attention to the details of postoperative care will go far in preventing postoperative hyperthyroidism. The patient should be isolated in a quiet, air-cooled room and given morphine in sufficient amounts to insure rest and allay nervousness. Hyperpyrexia is combated by the use of ice bags to the head and body. Of more importance are the maintenance of water balance and a supply of glucose sufficient to meet the needs of a greatly increased metabolism. This is brought about by the daily intravenous administration of at least 4,000 c. c. of physiologic salt solution and 100 gm. of glucose which is continued until the patient is able to take and retain this amount by mouth. Lugol's solution, 30 drops a day, is added to the physiologic salt solution, or given by mouth.

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DIVERTICULUM OF THE URINARY BLADDER*

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Vesical diverticulum is by no means a new disease nor is it a rare finding in the male. Much has been written with regard to diagnosis and the many methods of treatment, which have been devised within the past few years. To keep the subject alive and, possibly to suggest some improvement on points in diagnosis and treatment was the inspiration which prompted this presentation.

ETIOLOGY

That herniation of the mucous membrane through the bladder musculature, usually in the locality of the trigone, resulting in the formation of one or more diverticula is a developmental defect, seems quite clear if we bear in mind that one large sac should take care of existing back pressure and obviate the presence of other coexisting sacs. Obstruction of the bladder neck, however, in some form, such as strictures, obstructing bars and hypertrophied prostates, is a constant finding as evidenced by the fact that the disease is very rare in the female and then associated with some sort of obstruction. Age is also an important factor and though the condition is reported in youth, we have failed to find a patient younger than 20 years in our series. There has been only one diverticulum of the female bladder recognized as such in our work.

SYMPTOMS

The most common complaint is that of urinary frequency and slow, sluggish stream. Unless there is an obstructing prostate from hypertrophy there is seldom complete retention of urine, but always a certain residuum. A symptom which we have often observed is that of allowing the patient to urinate, then to repeat the act in a short time, when as much or more urine is voided at the second urination. This finding we attempt to explain on the basis of contracture of the diverticular neck at the time of voiding which later relaxes, allowing the contents of the sac to empty into the bladder.

Hematuria may exist, and is usually due to the presence of tumor, congested prostate or a ruptured blood vessel. One of our cases, in which the prostate was resected and a small diverticulum left alone, had periodic hemorrhages for which we accounted by the rupture of a vessel within the diverticulum. Subsequent observations proved the correctness of our assumption.

Very large diverticula may be palpated in the suprapubic area by the afflicted individual. Irregularity in outline is the rule and upon a few occasions the referring physicians with limited experience in such cases, have suspected the true condition. Infection is probably secondary, but usually exists, though one of our largest had no infection. It may be associated with chills and fever. As a whole, the symptoms elicited do not differ materially from those associated with any obstructive lesion of the bladder neck, but do serve as a basis on which to work for ultimate complete diagnosis.

DIAGNOSIS

If it is possible to introduce a catheter or a cystoscope, there is probably no disease in which

Fig. 1

B. Bladder.
D. Small diverticulum which has remained stationary following relief of the bladder neck obstruction.

*Read in Section on Urology, Southern Medical Association, Thirtieth Annual Meeting, Baltimore, Maryland, November 17-20, 1936.

*From the Departments of Urology, St. Louis University School of Medicine; St. Anthony's, Alexian Brothers, St. Louis County, St. Louis City Sanitarium, Jewish and U. S. Marine Hospitals.

Fig. 2

A. Bladder in contraction with small diverticulum above.
B. Diverticulum which was necessarily resected.

it is so easy to make a diagnosis as that of bladder diverticulum. Visualization of an opening, beyond which is a cavity, during a cystoscopic examination is sufficient indication for taking a cystogram. The technic of cystography is quite simple and can be done by an intern or well trained nurse. It consists of first, an x-ray exposure before injecting the bladder with 5 per cent iodide solution to the point of fullness, experienced by the patient, when a second exposure is made. We occasionally make a lateral view and many times have found stereoscopic views quite informative. Air contrast cystograms are valuable provided the patient is one who can withstand extra manipulation, though aqueous solutions usually fulfill all requirements.

Yet, with all of our improved technic and instruments of precision, we have seen this simple condition overlooked even when the bladder was opened. In fact, one of the largest diverticula in our series of 42 cases was missed at cystoscopic examination and at the first stage cystotomy incision for the removal of a very large prostate. There are no alibis to offer for such an oversight, except that the opening was immediately posterior to a very large, protruding median lobe and adjacent to the vesical orifice. It was easily palpated after removal of the gland and could have been as easily located if a cystogram had been made.

As stated elsewhere in the beginning, these sacculations may be single or multiple. All are demonstrated as to size, shape and position by cystography. Small and multiple buds have been shown by Herbst as the result of prolonged urinary difficulty, such as a slowly developing prostate hypertrophy. Naturally, the purpose of accurate diagnosis is to determine the method of procedure for complete relief if possible. Suffice it to say that vesical diverticula are no longer diagnostic or surgical curiosities.

ALLIED CONDITIONS

Since a diverticulum is aggravated and increased in size by the presence of some sort of vesical neck obstruction, we should expect many complicating conditions to exist. Probably one of the most common is peri-diverticulitis, where infection extends beyond the walls of the diverticulum causing a series of symptoms which are not suspected until the diagnosis of diverticulum is made.

Dilatation of one or both ureters frequently occurs and the opening of the ureter into the sac has been reported by other writers, though we have never found it. We have, however, found the ureter so intimately connected with the sac,

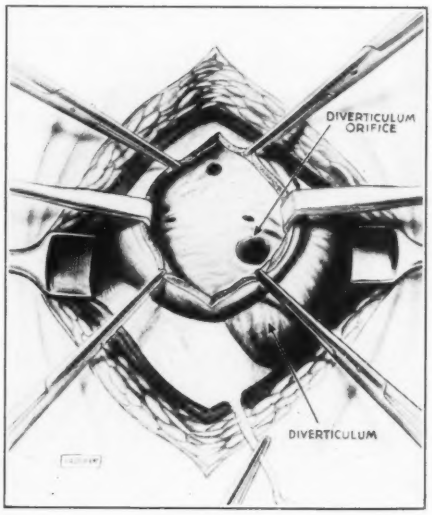

Fig. 3

Fig. 4

and the uretero-vesical opening so near the opening of the diverticulum, that it required very painstaking dissection to preserve it.

Small stones have been found a number of times in our cases and in one case a large stone. Two cases have had gonorrhea from which they eventually recovered. We have recognized one leukoplakia, but found no large tumors within the cavity of a true diverticulum.

TREATMENT

Inasmuch as diverticula are associated with obstruction to outflow, and are unquestionably harborers of retained urine, infection and their sequelae, the treatment of such conditions should be directed to correction of the aggravating cause and removal of the sacculation.

That correction of the obstruction should always be accomplished is, of course, beyond question. The mode of procedure varies with the type of obstruction present and the physical condition of the patient. Strictures and contractures which yield to dilatation are treated in the usual ambulatory manner. Median bars and obstructing lobes may be corrected by the punch method, incision or with any one of the many types of resecting instruments. True prostatic hypertrophy and carcinoma of the prostate require resection or complete enucleation.

Removal of a diverticulum is a problem that some time or other every urologist has to solve. It is a mooted question whether or not we are justified in submitting the patient, suffering with one or more small diverticula and an associated easily correctable contracture, to open operation. Our observations over a number of years have taught us that if the obstruction is corrected, in all likelihood the diverticulum will not materially increase in size. Moreover, these patients will develop immunity to the ever-existing infection and will probably be free from the aggravating dysuria with very little office treatment. There are, however, exceptional cases in which it is necessary to remove the sacculation in order to achieve the desired results. One of our most stubborn cases, of a total of seven, in which we have done a punch or resection and left unmolested a fair sized diverticulum has, after four years, become symptom-free. Others where only sounds or a Kollman dilator have been used, remain stationary with no complaints.

Large diverticula one-third the size of the bladder and frequently much larger than the bladder, containing foul urine, stones or other foreign bodies, require open surgical intervention. From a review of the literature it seems that these pathological conditions offered many problems to surgeons in the past. Of note are such procedures as inverting the sac into the bladder, excising the neck of the sac, ligating and allowing it to collapse, incising the neck and resecting the mucous membrane of the di-

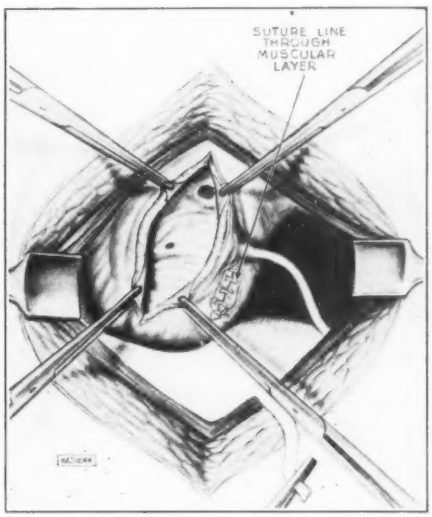

Fig. 5

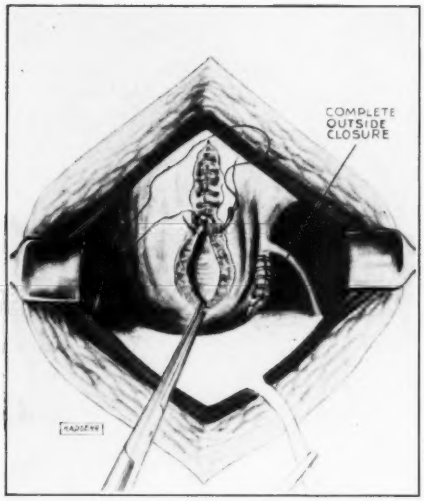

Fig. 6

verticulum, packing with gauze and complete enucleation of the sac outside of the bladder. There have been many other suggested procedures, but for the sake of brevity they will not be discussed.

We have tried most of the operations which have been suggested. With improvement of spinal anesthesia to the point of operative safety, it is possible to obtain excellent exposure of all the diseased parts when the operator can work with reasonable speed. We seldom use more than 100 mg. of procaine hydrochloride intraspinally, and frequently less than that amount, to resect one or more of the very largest diverticula. Proper postoperative care will insure good results in the most difficult case provided one is careful to withhold operation until the patient is in the best possible preoperative condition.

The method used in the last 8 cases is that of a long midline suprapubic incision, opening the bladder and palpating the diverticular orifice. With a finger in the diverticulum, used as a guide, not unlike that in a hernial sac, the diverticulum is carefully freed of all adhesions by means of blunt and sharp dissection. Occasionally it is necessary to cut the fibrous urachus for satisfactory exposure. We usually open the sac, though such a procedure depends upon the size. Eventually, after completely freeing all adhesions and assuring ourselves of the location and patency of the ureter and vas, the neck of the sac is excised to free muscle tissue, the mucous membrane of the neck cut away, and the opening closed in layers with No. 1 chromic catgut. Finally, the serous coat is closed tightly over the sutured muscle to insure against leakage. We have found it advantageous to drain the bladder in all cases through a suprapubic tube, owing to existing infection. It is also wise to drain the perivesical space by means of a cigarette drain through a stab wound of the lateral abdominal wall. This, however, though a simple procedure, is not obligatory. If a large prostate is present, diverticulectomy should be done at the time of the first stage drainage. Median bars and contractures, as well as small median lobes, may be removed with a Young's cold punch introduced through the urethra. In fact, this technic, with a finger in the bladder guiding the instrument, is carried out with greater ease than transurethral punch within a closed bladder.

ANALYSIS AND CONCLUSIONS

Over a period of 16 years, during which approximately 7,500 urological cases have been personally observed, about 30 per cent females and 70 per cent males, 42 cases of diverticulum of the bladder have been recognized. Of these, the youngest was 20 years of age. This is due, no doubt, to the fact that our hospital connections have never included a large pediatric clinic. There has been only one female. All have been white, which is explained by the fact that only a few colored charity patients enter into the group. The occurence of complicating or caus-

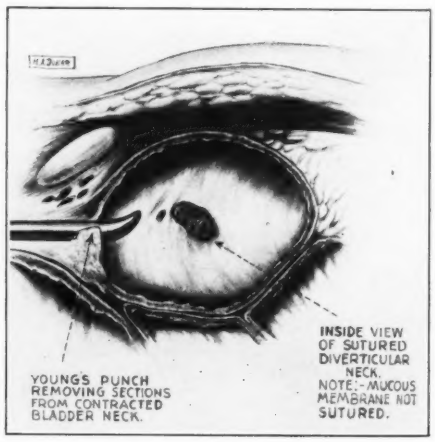

Fig. 7

Fig. 8

ative prostatic hypertrophies as compared with contracted bladder necks and median bars are approximately equal. Location of the diverticular openings shows a marked preponderance to the base of the bladder near the ureteral openings. Ten have been lateral and one definitely anterior. The sacs have never contained a large tumor. In no case has the ureter been found entering into the periphery of the sac though several times the ureteral opening and the opening of the diverticulum were closely connected. It has never been necessary to transplant or cut the ureter. There has been only one recurrence following complete diverticulectomy. Functional results have all been good in so far as it is possible to follow the cases operated upon. There have been two deaths, one this past summer during the extreme heat, and one of coronary thrombosis in which only the mucous membrane of the sac was removed at the time of complete enucleation of the prostate.

From an evaluation of this brief analysis it is apparent that the following conclusions are justified:

(1) In addition to the indisputable theory of congenital tendency to urinary diverticulosis, the exciting causative factors are contracted bladder necks and hypertrophy of the prostate. Statistics of individual physicians may vary according to the type of work done.

(2) The diagnosis is comparatively simple if routine cystoscopic and cystographic examinations are made.

(3) Small buds, single or multiple, will probably remain stationary if the obstructing lesion is removed by transurethral methods. If, however, the bladder is opened by suprapubic incision it is advisable to remove all pockets at the same sitting.

(4) Large diverticula should certainly be removed. The most satisfactory method is complete external excision working through the opened bladder with a finger inserted into the collapsed sac as a guide (see illustrations).

(5) Spinal anesthesia in moderate sized doses, seldom exceeding 100 mg. of procaine hydrochloride, has been of material benefit in allowing complete relaxation and ample exposure of the operative field. In careful hands and with average good fortune spinal anesthesia is the safest of all anesthetic agents in bladder surgery.

(6) Ample drainage of the bladder and all dependent pockets will reduce morbidity and mortality.

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DISCUSSION (*Abstract*)

Dr. James L. Estes, Tampa, Fla.—I wish to add another case to the literature of the rather uncommon condition, diverticulum of the bladder with a tumor in the base, with a distinctly different type of tumor in the kidney on the same side. The former was diagnosed preoperatively, the latter at autopsy. A man of 70 consulted me in February, 1935, because of frequency and burning on urination, accompanied by a strong ammoniacal odor to the urine. He had had pyuria for the preceding twenty-five years. He had been told that he had several small diverticula in the bladder, but he was unwilling to do anything about it, and refused to be cystoscoped. Rectal examination revealed only a moderately smooth, symmetrically enlarged prostate, grade 2. No induration or adhesions could be felt. He was put on large doses of hexamethylamine with bladder lavage. Five months later he came in passing large quantities of blood, for the first time. He entered the hospital for urological study. The kidney shadows were normal; there were no stones. Pyeloureterograms were normal. Blood urea was normal and kidney specimen clear. Cystoscopic inspection revealed several small diverticula in the bladder and a large diverticulum with opening located above and lateral to the left urethral orifice. The opening appeared to be about 2 cm. in diameter and the diverticulum quite deep. He was not bleeding at this time, and no place where the bleeding could be coming from was seen at this time, as I was doing a local cystoscopy and did not prolong it.

An immediate cystogram revealed the condition described above and the filling defect. Four days later he was again cystoscoped, and by advancing the beak far into the neck of this diverticulum I could easily see a flat, dark tumor, about 3 x 4 cm., which had been bleeding, but which was not bleeding at that time. After some delay and a confirmation, his bladder was opened and the diverticulum was removed by Dr. Leon Herman. The diverticulum was mobilized with some difficulty, but it easily tore loose at the base and left the tumor firmly adherent to the lateral pelvic wall. It was so extensive and so near blood vessels that very little was done. The perivesical area was packed and the bladder was closed. After a stormy course he recovered from operation. He received a large amount of deep irradiation for the next 4 months. For 6 months after operation he passed no blood, but about the seventh month the tumor had reinvaded the bladder and the urine was mostly blood. About this time he developed a complete left-sided paralysis and vomited continuously until death 9 months after the operation.

A partial autopsy, with removal of tissue, showed the tumor to be about 5 times the original size and that it had invaded all of the surrounding structures. In addition, the left kidney, which was small, showed a tumor 3 x 4 cm. in the lower pole.

Sections from the extra-vesicular mass were definitely malignant and showed small infiltrating cords and columns of undifferentiated carcinomatous cells surrounded with a fibrous stroma. In some areas, the picture was much more cellular, showing solid masses of tumor cells without papillary arrangement, with marked variation in size, shape and staining.

Sections from the intra-vesicular tumor were essentially like the above, except that the scirrhous picture predominated.

Sections from the kidney tumor showed no relationship with that of the bladder. The picture is that of a congerie of cylindrical columns of large clear cells, surrounded with thin bands of fibrous stroma. The sections were very vascular and contained many blood sinuses.

The pathologic diagnosis by Dr. H. R. Mills was carcinoma of the bladder; adenocarcinoma of the kidney with large clear cells (so-called hypernephroma).

Although no autopsy was done on the brain, I feel certain that he had metastasis in the brain.

RECENT DEVELOPMENTS IN THE CINEMATOGRAPHY OF THE FLUOROSCOPIC IMAGE*

By WILLIAM H. STEWART, M.D.
and
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By cine-fluorography is meant the making of a motion picture record of the image seen on the fluoroscopic screen. Attempts to accomplish this purpose were begun soon after the discovery of the roentgen ray and have persisted at intervals ever since.

The difficulties surrounding this procedure have been greatly lessened by new developments in apparatus recently made available. The apparatus necessary for cine-fluorography is briefly described.

Camera.—An ordinary Bell & Howell filmo 70-D 16 mm. camera was used fitted with a special view finder due to the large size lens used.

Lens.—A Zeiss R-biotar with an aperture of 0.85 and a focal length of 5.5 cm. was employed.

In order to cover the field of the fluoroscopic screen it was necessary to use a camera-screen

*Read in General Clinical Session, Southern Medical Association, Thirtieth Annual Meeting, Baltimore, Maryland, November 17-20, 1936.

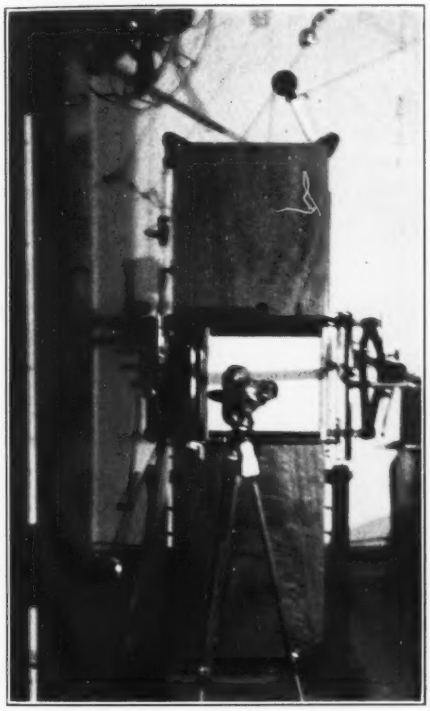

Fig. 1
Special screen, camera and lens used in cine-fluorography.

distance of seven feet. The lens is employed wide open.

Film.—Best results in our experiments were with Eastman panchromatic super-x film.

Fluoroscopic Screen.—The brilliance of the fluoroscopic image varies with the chemical composition of the screen, while the photographic result depends upon the sensitivity of the film to that particular wave length of light emitted by the screen. We have found that the most desirable combination is a special cyanide yellow green screen used in combination with Eastman panchromatic super-x film. This screen is made from a preparation of platinocyanide developed by Dr. Leonard Levy and Mr. Donald W. West of London.

X-Ray Tube.—The Philips mammoth 20 K.W. tube was operated at practically full capacity. This exposure is well within the limits of safety for the apparatus and the skin tolerance of the patient.

If the camera is used at sixteen frames per second, thirty-two pictures are obtained in the exposure time of two seconds. This is sufficient to record motion of the heart, lungs and other rapid movements in the viscera. This piece of film can be made into a loop and continuously projected for study as long as the observer pleases.

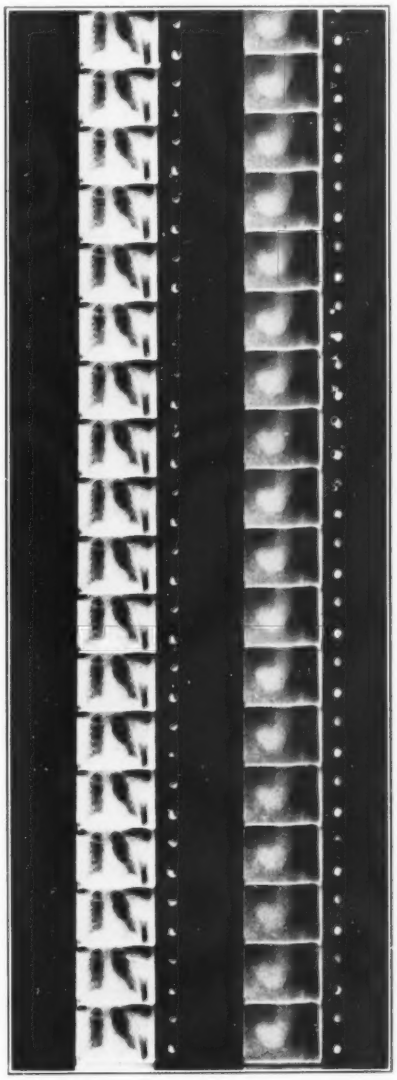

Fig. 2
Normal chest. Normal stomach in diastole.

Films made at this speed and projected reveal motion at a normal rate.

If the patient is unusually thick it may be necessary to slow the camera speed to twelve or even eight frames per second so that an x-ray exposure twice as long may result. The motion of the organs is then twice as fast. In thin patients the camera speed may be increased to twenty-four pictures per second, so that when the film is projected the motion is slowed down, permitting detailed analysis of any part of the moving organ, for example, study of the contractions of any of the chambers of the heart.

Technic.—The camera is placed on a tripod at seven-foot distance from the fluoroscopic screen. The patient is placed as usual for the fluoroscope and the screen is adjusted to demonstrate properly the field being photographed. The tripod is regulated so that the camera is centered. A trial exposure at a low fluoroscopic setting is made to test for correct position. The two-second exposure is then made with 100 KV. and 110 MA.

Previous to this time the difficulties with the method have been insufficient brilliance of the fluoroscopic image to permit perfect photographic detail. This has been solved by the above technic and apparatus for the thorax in almost any size individual.

Below the diaphragm, however, the body is less easily penetrated by the x-ray and to date it has been possible to photograph satisfactorily the abdominal viscera only in thin, favorable subjects. Gastric and duodenal lesions are demonstrated, but the exposure in many cases is too short to record the entire physiological cycle of activity of the stomach and duodenum. This we hope to overcome by the use of a synchronizing switch which allows the x-ray beam to reach the patient only when the shutter of the camera is open.

The practical usefulness of the method may be summarized as follows:

Many moving organs may be intensively studied at leisure as long as desired without exposure of the patient or operator to the x-ray.

In order to study heart action heretofore it has been necessary to sit at a fluoroscope exposed to x-ray and make momentary observations.

With a cine-fluoroscopic film one may see more clearly than possible with any fluoroscope the heart motions which may be studied as long as desired. It has been necessary heretofore for each and every observer to do his own fluoroscoping and record his individual observations. With this new method the film may be observed by a hundred or more and it forms a permanent record which may be consulted anywhere at any time. Its advantages for teaching are thus manifold.

Almost all heart lesions are better demonstrated when study is made of the moving organs rather than the flat roentgenograms. Pericardial effusion is differentiated from decompensation; aortic aneurysm from mediastinal tumor; the motion of fluid within cavities can be observed; the extent of the collapsed lung in pneumothorax can be recorded; the degree of mediastinal shift in pneumothorax; the range of the diaphragmatic excursion; the common lesions of the esophagus; joints with partial limitation of motion and other important problems formerly capable only of individual interpretation fluoroscopically may now be studied intensively by groups if desired.

The motion picture is even clearer than can be seen on the best possible fluoroscopic screen because more current is used in order to photograph the fluoroscopic image.

The method is more economical than using x-ray film and may prove of practical importance from this aspect alone.

It seems likely that this new procedure is destined to become an important division of roentgenologic investigation.

LUPUS VULGARIS IN THE FAR SOUTH*

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and
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The influence of climate upon the incidence of tuberculosis of the skin is generally recognized.

Frequent in north and central Europe in a cold, humid climate, deficient in sunlight, cutaneous manifestations of tuberculosis become less frequent as one approaches the equator, being rare in Italy, Spain, and North Africa.

By comparison with Europe cutaneous tuber-

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*From the Dermatological Department, Jackson Memorial Hospital, service of Dr. Elmo D. French.

culosis is rare in the Americas, again becoming even less frequent as one approaches the equator.

No proven case of lupus vulgaris originating in the State of Florida, it is believed, has ever been reported. In passing, however, it can be said that other forms of cutaneous tuberculosis have been observed.

Situated upon the Atlantic Seaboard, latitude 25° 48′ N., longitude 80° 12′ W., the Miami area has a mean 25-year average temperature during the five winter months of about 70° F. The air is free of pollution, with prevailing winds from the east and southeast passing over the gulf stream. During these same months there is an average of 1,081 actual hours of sunshine which is reflected from ocean water, sandy soil and the lightly tinted stuccoed walls which prevail in buildings.[1]

This compares with an average mean temperature of 38° F. and 810 actual hours of sunshine for Washington, District of Columbia, and a mean temperature of 47° and 725 actual hours of sunshine for Chicago, during a like period of time.[1]

The following case to be reported was brought into the Miami area and it was thought that observations upon the course of the disease in an environment unfavorable to cutaneous tuberculosis would prove of interest.

B. R., a man 29 years of age, married, with no children, was admitted to the hospital April 14, 1936. He was born in Chicago, his parents being Jewish immigrants from Russia. His father was his mother's uncle, she having married her mother's half-brother. One sister by this marriage is living and in good health. A half-sister, by a previous marriage of his mother, has convulsive attacks followed by loss of consciousness.

The father is living and well at age 60; the mother met with accidental death.

There is no history of tuberculosis in the family nor has the patient ever knowingly lived in a tuberculous environment.

He was a full term infant who weighed 10 pounds at birth, and was breast fed. The first two years of his life were uneventful and development was normal.

At the age of 2 years his left foot was operated upon by the late Dr. John B. Murphy for bone infection. The site of operation did not completely heal, draining pus until he was 7 years of age, or five years after operation.

It was shortly after this operation that skin lesions began to appear on various parts of the neck, trunk and extremities, beginning as small papules which spread slowly outward from the center.

When he was 5 years of age, several inches of the distal end of the left radius were removed. Healing

Fig. 1

Ulcerations and erosions at the center of a lupus; the latter involving a large portion of the posterior plantar region, heel and lateral side of the left foot.

Fig. 2

Lupus vulgaris in varying stages of evolution. An isolated lupoma on the lateral surface of the upper lip, a group of lupomas of the lower nose, a lupus plaque extending from the outer margin of the eye-brow upward and backward on the temple and an extensive lesion of the neck.

Fig. 3
Lupus vulgaris of the scalp: An extremely rare lesion especially when occurring isolated. Final resolution resulted in a porcelain white cicatrix.

was uneventful and the discharge of pus, which preceded operation, did not recur.

There was an attack of measles at 6 years of age, which had no noticeable effect upon the course of his skin affection.

During the twenty-seven years' duration of the disease, periods when new lesions appeared were followed by periods of regression when the existing lesions appeared to stop extending and began to form scars, but later they again extended into previously sound skin.

Local measures for relief in various hands have run the gamut of roentgen ray treatment, artificial ultraviolet treatment from both mercury and carbon lamps, endothermy, curettage, and caustic pastes with either temporary, indifferent, or no success.

The Gerson-Hermansdorfer Sauerbruch diet was attempted without hospital supervision for several months and its failure under such conditions might have been anticipated.

Intravenous administration and local injection of a gold salt resulted in no improvement.

Up to the time of admission to the Jackson Memorial Hospital neither tuberculinotherapy nor heliotherapy had been attempted.

A well-nourished, medium-sized man of robust physique, weighing 139 pounds, presented multiple lesions of the skin and mucous membranes, involving the scalp, face, neck, trunk, the upper and lower extremities and buccal and nasal mucosa.

The skin lesions varied in size, depending upon the age of the lesion, from single papules to confluent papules forming more or less extensive plaques, the largest of which encircled three-fourths of the neck, extending downward for a varying extent upon the upper thorax.

The lesions likewise varied in aspect, according to anatomical site. A lesion of the left hand caused marked cicatricial contraction, one on the left foot was ulcerated, one on the vertex of the scalp was a smooth, slightly elevated scar with a narrow peripheral red zone. The majority of lesions, however, were varying sized plaques, on the whole, typical of those described as the flat non-ulcerative type of lupus vulgaris; red violaceous in color, slightly infiltrated with denticulate or irregular borders and usually lupomas or "apple jelly nodules" at the periphery. Squamae either did not exist, when the surface was smooth and glistening, or in places, as the left elbow and plantar surface of the right foot, were abundant, approaching psoriasiform character. Slight erosion of the surface likewise occurred in these latter lesions.

New lesions were appearing beginning as small pinhead to split pea sized, smooth, dark red papules. These were isolated, grouped, or confluent and were evident upon the lower one-half of the nose, anterior fold of the left axilla and posteriorly over the right scapula, below the left scapula and at the root of the left thigh anteriorly.

The mucous membrane of the cartilaginous portion of the nasal septum showed several distinct pin-head, reddish yellow, slightly elevated papules.

The mucosa of the hard palate presented a central erosion with erythematous borders, ragged in outline.

The lower one-third of the left radius was missing and there were deeply depressed scars in the lateral and planter surfaces of the left foot, appearing as healed sinuses.

Fig. 4
(Part of the distortion at the wrist is due to the removal of the lower end of the radius when 6 years of age.) Marked cicatricial contraction and squamous alteration.

Fig. 5

Lesion of elbow shows marked degree of epidermal alteration, described as psoriasiform lupus vulgaris. Lupus nodules active in the center of the cicatrix of the forearm lesion.

The superficial lymph nodes were palpably enlarged, fairly hard, painless and discrete, especially the superior and inferior inguinal nodes.

Chest expansion was free and equal on both sides, resonant throughout and breath sounds were unaltered.

The heart was normal in size, shape and position, regular in rhythm, with no murmurs.

The abdomen was flaccid, with no masses and no tenderness.

The genitals were normal in appearance.

The red blood count was 4,170,000; hemoglobin, 92 per cent; white cells, 6,800; differential, segmented 56; lymphocytes, 32; monocytes, 4; eosinophils, 8.

The Kahn test was negative.

The sputum was repeatedly negative for tubercle bacilli.

The urine was chemically and microscopically negative. A concentrated specimen was stained for acid fast bacilli, which were not found.

The Mantoux test was done, utilizing old tuberculin in a dilution of 1-100,000. There resulted in 48 hours a dime-sized nodule surrounded by an erythema. The lupus lesions became a more vivid red, were turgid, the ulcer on the left foot became painful and edematous with increased exudation, a strongly positive local and focal reaction.

Radiologic Examination (Dr. A. G. Levin).—"Examination of the chest shows the lung fields and costophrenic angles to be clear. There is no evidence of old or recent parenchymal infiltration. The heart is within normal limits in size and contour.

"Examination of the left foot shows irregularity of the margins of almost all of the tarsal bones, particularly the navicular and cuboid and the anterior portions of talus and calcis. The joint spaces between these bones are widened. The navicular appears considerably diminished in size and increased in density, particularly in comparison with the opposite side, which appears normal. There is no evidence of new bone formation at the margins of the involved bones nor is there any evidence of bony ankylosis. There is considerable atrophy of the soft parts. The findings suggest an old inflammatory process, but are not typical of tuberculosis."

Histo-pathological Examination.—Biopsies were taken, April 16, from an early lesion of the posterior left shoulder, the lesion being removed in its entirety and a second biopsy at the margin of an old lesion on the left arm.

Microscopic examinations were reported as follows, by Dr. A. G. Levin:

"Sections from one of the early lesions on the shoulder show the surface epithelium to be flattened and in some areas partially atrophic. The corium is very heavily infiltrated with round cells and epithelioid cells, many of which are arranged in the form of tubercles with associated giant cells of the Langhan's type. There is very little necrosis.

"Sections from one of the older lesions on the arm show similar changes, except there is more marked damage to the surface epithelium in some areas and a few small areas of necrosis are present."

Attempts to demonstrate acid-fast bacilli in sections of this material were not successful.

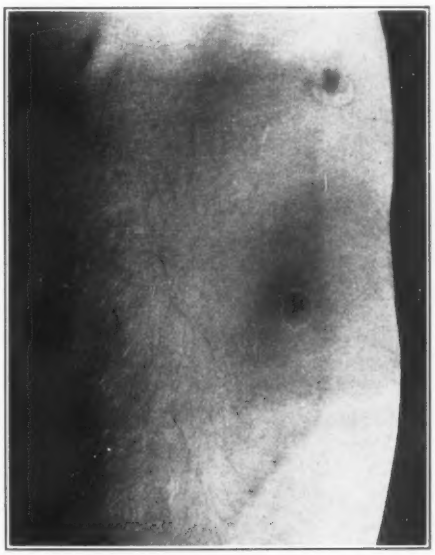

Fig. 6

Positive Mantoux test photographed 48 hours after injection of "old" tuberculin in 1-100,000 dilution.

Fig. 7

Section from the thigh, showing tuberculous reaction in papillary and upper reticular portion of the corium, consisting of nests of epitheloid cells with peripheral zone of lymphocytes and plasmocytes and occasional giant cell within the nests. (Hematoxylin and eosin).

On April 29, under careful aseptic precautions, an early lesion was removed entirely from the region below the left scapula.

With this material so obtained three guinea pigs were inoculated. One beneath the right and left rectus fascia, another deep into the thigh muscles and the third intraperitoneally.

The first two pigs remained healthy. The third died July 9 between nine and ten weeks after inoculation. There was marked loss in weight. The spleen and liver were studded with miliary abscesses. A retroperitoneal lymph node was selected from the sacro-iliac region, about the size of a pea and containing caseous material. Stained smears showed acid-fast bacilli.

Half of this node was injected subcutaneously beneath the rectus sheath of a rabbit. A nodule containing caseous material formed at the site of injection, but the rabbit remains well to date after four months.

ETIOLOGY AND PATHOGENICITY

Pautrier[2] says: "Other than atypical cutaneous tuberculosis and the tuberculids which are due to a single mode of infection, there is not any form of classic cutaneous tuberculosis which is due to a single and unique mode of infection, and no mode of infection always produces the same clinical lesion."

The history of this case clearly indicates that the first known site of tuberculosis occurred in the small bones of the left foot and the primary lesion of lupus vulgaris occurred as an extension from this focus following operation.

The subsequent clinical history of the disease, with the appearance, throughout its entire course, of new lesions, which were noted upon first examination, would seem to make their hematogenous origin indisputable.

Since we lack evidence of active visceral or bony foci of infection at the present time, the following quotation from Adamson[3] is interesting:

"There is evidence, as has been shown by Gridlestone, that these metastatic lesions in bones and viscera are not themselves sources of further metastatic infections, that tuberculous visceral and bone lesions do not and cannot themselves give rise to bacillemia, but that this must always arise from lymphatic foci."

Experimental evidence adduced would indicate that the infecting organism was of the human type, inasmuch as authorities agree as to the very low susceptibility of the rabbit to the human strain of tubercle bacillus, while by contrast it is very highly succeptible to the bovine strain.[4 5]

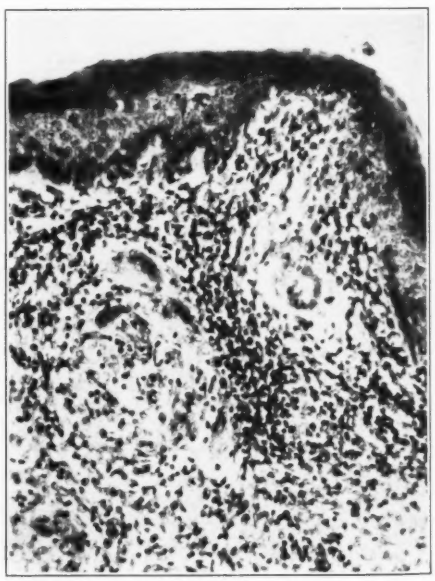

Fig. 8

High magnification of subepidermal nests seen in Fig. 11.

Fig. 9

Section from the thigh, stained with acid orcein, showing almost complete destruction of the elastic tissue in the infiltrated areas.

A test rabbit is living and well four months following inoculation with material proven to contain numerous acid fast bacilli, the virulence of these organisms, originating in a cutaneous lesion of the patient, having been increased by transmission through a guinea pig.

Local Treatment.—Complete ablation of five recent lesions was performed. These lesions varied from groups of several pea-sized lupomas up to small coin-sized plaques.

No attempt at radical destruction was made upon the remainder of the lesions except the intractible, painful ulceration of the left foot.

Attempts to destroy this ulcer with 30 per cent zinc chloride in 90 per cent alcohol failed, as did likewise the use of insulin 10 per cent in lanolin, of which cicatrizing effect has been testified to by Pautrier[6] and others. Recently Throne and Myers[7] reported upon the use of this agent in the treatment of non-diabetic varicose ulcers. It is interesting that our experience in a tuberculous ulcer was similar to their own, that is, there was rapid temporary improvement followed by aggravation.

Destruction of the ulcer with endothermy followed by curettage down to the plantar fascia and subsequent application of silver nitrate 1 per cent, baisam of Peru 5 per cent, in lano-vaseline, resulted in epithelialization.

General Treatment.—Cod liver oil and di-calcium phosphate were administered by mouth.

Except for a liberal allowance of green vegetables with fruits and legumes, no especial dietetic modifications were undertaken.

Heliotherapy was begun soon after admission, beginning with 15-minute exposures, which were rapidly increased to exposures of one and one-half hours.

There were no special refinements in method of administration.

Old tuberculin was administered intradermally in amounts of 0.1 c. c. as follows:

April 20, 1936	1-100,000
April 30, 1936	1-10,000
May 10, 1936	1-5,000
May 21, 1936	1-5,000
May 26, 1936	1-5,000
June 1, 1936	1-5,000
June 8, 1936	1-5,000

Following each injection within 48 hours there was a focal flare in all the lesions with edema especially noted at the periphery where the capillary circulation was most intact.

This suggested that Moro's ointment containing 10 per cent old tuberculin in lanolin (not ektebin) rubbed directly into the lesion would be more effectual, which proved to be true.

The lesion so treated became more edematous than the ones at a distance and this was followed by correspondingly greater regression of the lesion. Also an application of this ointment to a posterior portion of the neck lesion showed a more marked reaction and subsequent regression in the area where the ointment was applied, by comparison with the remainder of the same lesion.

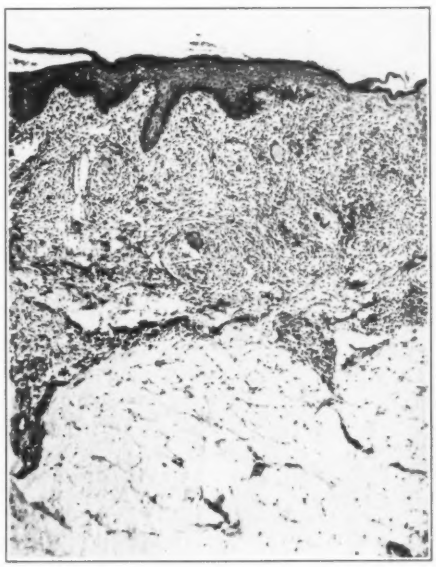

Fig. 10

Section from the shoulder, showing classical tuberculous architecture involving the upper half or two-thirds of the corium. (Hematoxylin and eosin).

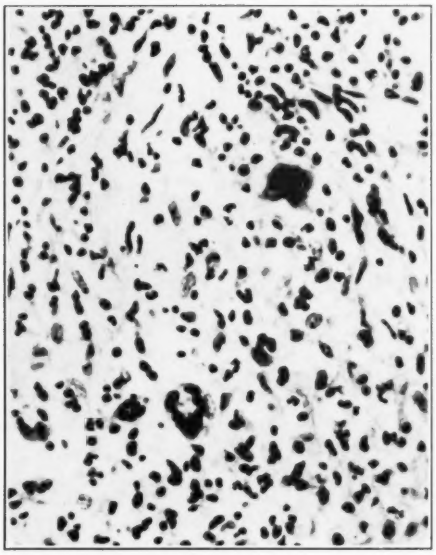

Fig. 11
Section from lymph node of infected guinea pig, showing tubercle formation. (Hematoxylin and eosin).

A pea-sized mass of the ointment was used, no single lesion receiving more than one application, at intervals as follows: June 24, June 29, July 11, July 24, August 22, and August 28.

Results of Treatment.—During the first six months in the hospital the patient gained pounds in weight. There has been a further increase in weight, the patient now weighing 162 pounds.

The occurrence of new tuberculous lesions, noted for a short time after admission, ceased.

The more recent tubercles are now either not distinguishable in the surrounding deeply pigmented skin, or like those observed on the nose and left temple and at the periphery of the lupus plaques, they appear as slightly darker macules.

The lupus plaques are no longer so sharply limited in outline; their centers are irregularly pigmented on a white sclero-atrophic background, presenting in places a "piebald" appearance. The erythematous color is almost wholly lacking in these lesions and they are scarcely elevated, or on a level with the surrounding skin. There is perceptible softening with less cicatricial contraction of the lesions, allowing the patient more mobility where the flexures are involved.

The ulceration, several years in duration, of the left foot has completely epithelialized.

A slightly dark discoloration marks the site where the hard palate was eroded. The lupus nodules in the nasal septum are scarcely perceptible.

COMMENT ON HELIOTHERAPY

The role of the skin as an organ of defense was first termed "esophylaxis" by Hoffman[8] and later "dermophylaxia" by P. Worringer.[9]

This function is awakened, re-enforced or stimulated by the actinic, ultraviolet rays of the solar light.

The function is non-specific and acts in the same fashion upon all infectious processes which attain maximum intensity in the winter and spring, among these, tuberculosis.

Again the vitamin deficiency consequent upon a paucity of biologically active radiation, especially observed following the winter months, and its effect upon immunity and metabolism, is admitted.

Solar radiation, while not giving rise to specific antibodies, can at least be said, then, to increase general antibody formation by its general beneficial effects.

In addition to the general effects of solar radiation, however, the local effect upon cutaneous tuberculosis is not considered negligible and the case here presented seemed to adapt itself particularly well to heliotherapy.

Coblentz[10] estimates the probable depth of penetration of near ultraviolet, 2,900 to 3,650 Angstrom units at 0.5 to 1 mm.

In view of the well known sensitiveness of the bacillus of Koch to ultraviolet, two hours of irradiation sufficing to sterilize cultures of this organism, according to Pautrier, it may be assumed that inasmuch as in the case reported the infiltration did not extend to great depth, the ultraviolet rays were able to exert a maximum local effect upon the tubercle bacillus *in vivo*.

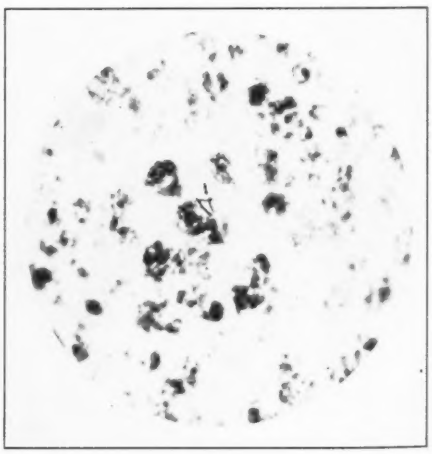

Fig. 12
Smear from lymph nodes of infected guinea pig, showing tubercle bacilli. (Ziehl-Neelsen stain).

SUMMARY

(1) The rarety of cutaneous tuberculosis as one approaches the tropics is discussed.

(2) Climatic conditions favorable or unfavorable to the disease are compared.

(3) A case of multiple lupus vulgaris transported to south Florida is presented, with positive proof of the diagnosis by animal inoculations of diseased tissue.

(4) Comments are made upon the pathogenicity of this case, including reasons for considering the lesions to be of hematogenous dissemination and the infecting tuberculous organism to be of the human type.

(5) The treatment especially emphasizing tuberculinotherapy and heliotherapy is outlined.

(6) The subsequent favorable course of the disease is described.

(7) Comment is made upon the general and local effects of heliotherapy.

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DISCUSSION (*Abstract*)

Dr. J. Lee Kirby-Smith, Jacksonville, Fla.—During twenty-six years of practice of dermatology in North Florida I have not observed a single case of primary tuberculosis cutis, that is, lupus vulgaris, or tuberculosis verrucosa cutis. Only rarely have I observed any secondary infections, for instance the scrofuloderma. As to the tuberculides, Bazin's disease, papulonecrotic tuberculide, sarcoid of Boeck or granuloma annulare, the cases seen by me in Florida are not more than twenty. Yes! Unquestionably a sunshiny climate has a great controlling influence on the incidence of tuberculosis and particularly on tuberculous infections of the skin.

Sunlight as a means of treatment of this disease, tuberculosis, was first used with success in the Alps at St. Moritz. In more recent years, ultraviolet rays obtained from a carbon arc light at Copenhagen and London have given us favorable results in the treatment of lupus vulgaris. During several months of the writer's attendance at the Light Department of the London Hospital many cases seen there had been practically cured by the Finsen light.

Today's report by Drs. French and Lefholz emphasizes my opening statements. The case they have presented is of unusual interest, unquestionably an extensive lupus vulgaris of a number of year's duration, originating in a northern state.

The results of the treatment of this patient are remarkable. The authors did not say how much was attributed directly to heliotherapy though unquestionably it helped to a great extent. However, the diet and tonics outlined, tuberculin injections, local measures, electro-surgical treatment, and the use of an ointment of tuberculin were all contributory. No one questions the extent of the clear and intense sunlight of the Miami district, even in the midst of winter. This is advertised far and near. It would not be amiss to say here that there is an unusual prevalence of lupus erythematosus both in South Florida and in North Florida, due to a great extent to undue sun exposure and in no way connected with tuberculosis.

Dr. William F. Spiller, Galveston, Texas.—Drs. French and Lefholz have presented a case which is of unusual interest from a standpoint of climatic effect upon lupus vulgaris. They have pointed out to us reasons why this disease is rarer in the southern coastal areas than in the northern sections.

They have given a definite proof as to the correct diagnosis in this case and, also, that it originated from bone tuberculosis of the foot.

Lupus vulgaris is claimed to be the most common type of cutaneous tuberculosis. However, I have never seen a case that originated in Texas. A few cases of other forms of cutaneous tuberculosis, which I knew originated in Texas, have come under my observation.

At the clinic presented at the Texas State Dermatological meeting in Galveston this month we had two cases of tuberculosis of the skin, one of which was lupus vulgaris. This case originated in another state. It was an old case, which was complicated by an epithelioma in the scar; the other case, I feel sure, originated in Galveston.

Dr. H. C. Sinderson (transactions Royal Society of Tropical Medicine and Hygiene, Vol. XXV, No. 1, June, 1931) said: "Lupus vulgaris is relatively uncommon in hot countries presumably on account of the inimical effect of sunlight on the causal organism." This is the contention of the essayist in this case.

I agree that the reason lupus vulgaris is rare in the South is that here we have more ultraviolet radiation in the solar light.

The etiologic organism is always the tubercle bacillus, the invasion being either primary or metastatic, although bacilli may not be easily demonstrated. It usually begins between the ages of 12 to 25, and is more prevalent in poor hygienic surroundings. In the *British Journal of Dermatology and Syphilology* in 1931, Dr. W. J. O'Donovan reported several cases of primary lupus vulgaris in infants. Ravages wrought by this disease are often extensive and horrible.

Many methods have been advocated for the treatment of lupus vulgaris, among them x-ray, radium, Finsen light, diet and tuberculin injections. Treatment is not limited to any one method.

It is conceded that climate has a distinct beneficial

effect upon this disease, but alone it is not a cure, and the case reported supports this fact.

Dr. French (*closing*).—In a location where lupus vulgaris is not endemic, it would be obviously presumptuous for one to attempt evaluation of treatment in this disease.

We do feel, however, that the observations recorded in this case emphasize the beneficial effects of combined specific antigen and heliotherapy.

A warm climate allows exposure of the body to sunlight. As all dermatologists know, the face and extremities, which are subject to congestion in a frigid atmosphere, are sites of election for tuberculosis of the skin and tuberculids.

Climatology has not received the attention it deserves in medicine. A closer cooperation with physicists is desirable.

SUGGESTIONS AND INQUIRIES ON THE THERAPY OF SKIN DISEASES*

By M. Toulmin Gaines, M.D.
Mobile, Alabama

The presentation of this paper is not based on any assumption of superior knowledge on the part of the writer, but in acquiescence to the request of our chairman and our secretary, and in complete agreement with them as to the necessity of giving this subject more of the time and attention of our section.

The elimination of our round table discussions apparently carried away with it all interest in the cure of our patients; for I am sure, were it possible to have had stenographic records of our clinical discussions, we should find in them scant references to the treatment of the cases presented.

In the few other dermatological clinics that I have been fortunate enough to attend, each of us was provided with a complete history of the case and the treatment up to date. Yet the resulting discussions revolved about the subject of diagnosis, while the subject of treatment was either ignored or confined to a few casual remarks. I can recall one exception where the subject of the treatment of atopic eczema received a full discussion with a variety of helpful suggestions.

In the reports we read in the archives of dermatological meetings all over the country, the cases are almost always presented for diagnosis, while treatment is rarely mentioned.

Some of the text books devote pages to description, diagnosis, and etiology, and give a few short sentences to treatment.

The recent inclusion in the work of our good friends, Drs. Wise and Sulzberger, of a yearly article on treatment has received disapproving comment from the *Archives*, a criticism which they will do well to ignore.

At our recent meeting we heard one of our foremost men say that for years he had not been interested in curing people; but in trying to find out why they had these conditions. Of course, this is in line with the scientific attitude to which is due the advancement of our calling. Medicine recently has been accorded more and more acclaim by reason of the progress in preventive medicine making it possible for our complex civilization to carry on in comparative security.

But the unfortunate individuals should not be neglected. We are a body of physicians and should not let the scientific attitude entirely replace the humanitarian service on which our profession justly prides itself.

In our section clinics, why do we jostle and crowd one another and willingly spend untiring hours inspecting these cases, and then spend an entire evening in discussing their conditions, and yet are apparently indifferent as to their final well-being? How often do these unfortunates appeal to us for help, and as we rightly refer them to their physician, how rarely do we offer the physician in charge any suggestions, even though he repeatedly requests them? Is our scientific attitude the sufficient explanation or would it be amiss to indulge in a little introspection?

Is it egotism? Is it the pinning of our wits one against the other? Are the patients merely pawns to give us the chance to match our experience, or to compete in shrewdness of observation?

Is it diffidence? Is it the unwillingness to have our claims of therapeutic success met with incredulous tolerance; to hear again the courteously suggested charge that our conclusions are based on insufficient data?

Is it prudence, a hesitancy to display our most cherished methods lest we expose our limitations?

Is it the therapeutic nihilism so much in vogue with some general practitioners? If so, we should not be drawn into that attitude. Our branch is the best demonstration against the cultists. Not forgetting the neuropathic origin of some skin disorders, the fact remains

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that skin disease cannot be cured by denying their existence, by spinal adjustments, by interpretation of dreams. Who among us does not include, in his clientele, Christian Scientists, chiropractors, Holy Rollers, and so on?

In answer to the above queries the writer acknowledges the diffidence, having felt rather than heard the incredulity with which the claims of the clinician of moderate opportunities are received by those who can command the incommensurable figures of crowded clinics. But are not such figures of more value as to the incidence and proportion of various diseases, than as to the therapeutic results? Is not the value of dispensary therapeusis impaired by the routine formulary, hurried directions, follow-up failure, shiftless patients, lessened personal interest?

Dr. Benson Cannon has said that scattered all over the country are dermatologists of moderate practice who are good clinicians. Would not the sum total of their experience be of practical value? For as the years pass this sum total is not negligible even as compared to the crowded clinic. Nor is a limited environment without its compensation, for in the small town knowledge of everybody's business, his failures are noted and his successes are almost invariably reported to him.

I am further emboldened to forget my diffidence, and disregard the doubts of my hearers, and to throw away prudence and let you perceive my limitations, by recalling more than one instance in which patients from noted dermatologists have reported failure to be benefited by valuable prescriptions accompanied only by the direction "apply twice daily."

At the risk of being prolix and tedious with some repetition of what I have said once before and what has been written by my colleague, Dr. Cogburn, I shall describe in detail our painstaking and untiring treatment of two very common conditions with gratifying results.

Some years ago I heard a discussion in our round table on the treatment of the superimposed pus infection of epidermophytosis; the question being whether the ringworm should be treated from the outset or whether the infection should be first cleared up. One now highly honored by us confessed at that time to a vacillating course, trying for awhile the first method on his cases and, becoming dissatisfied, veering to the second horn of the dilemma on his next set of cases.

What I stated then in outline I shall now write in detail. The method is simplicity itself, but requires diligence in its performance. It consists in the continuous application of Thiersch's solution, which in addition to cleaning the parts, inhibiting the formation of pus, macerating the corneous layer, giving access to the pockets and undermined edges, is mildly antiparasitic. We modify the original formula of salicylic acid 2, boric acid 12, to water 1,000, by substituting for the water D. D. Hammamelis 250. The directions being "dilute with three parts of boiled water for continuous application."

The patient is instructed to boil about two quarts of water for ten minutes. With half of this scald out a quart milk bottle or fruit jar. Allow the other half to cool. Pour entire contents of prescription in the jar and fill with the boiled water.

Strips of the eightfolds of gauze are cut to fit around the fingers and hand or toes and feet, soaked in this solution, applied, covered with wax paper and held in place by just sufficient bandages. The patient is instructed to drip the solution at frequent intervals under the wax paper. He is to change the dressing night and morning, and if there is much discharge and debris, also at noon. The morning dressing is changed at our office, at which time the dead corneous layer is trimmed to prevent undermining of edges or forming of pockets. Two or at most three days are sufficient to remove the corneous covering and practically clean up the pus infection. If there is still some adherent corneous debris it can be removed by applying simple ointment of acid salicylic 2, vaseline 32. When the underlying surface is pink or denuded an x-ray exposure of one skin unit is given. The skin is then soothed with cold cream until it is sufficiently repaired to admit of the use of an ointment of salicylic acid 2, sulphur precipitate 4, thymol .26, lanoline 16, vaseline 16. This is to be used night and morning by thoroughly rubbing in. The patient reports on the eleventh day to note evidence of roentgen reaction. This application is kept up until the twenty-first day, when the x-ray exposure is repeated if there is any suggestion of resistant areas.

Often near the end of the twenty-one day period there will be a recurrence of the characteristic grouped vesicular eruption. The parts are then treated with the Whitfield ointment, acid salicyl 3, acid benzoic 4, lanoline 16, vaseline 16.

The method of applying this salve will be given in detail as upon carrying out these instructions absolutely, we consider the cure depends. The written directions on the prescription are merely "spread on cloth and apply twice daily." The patient is carefully instructed not to use gauze as in a single layer the interstices are so large that it will not hold sufficient salve, while in more than one layer the gauze will absorb the salve and decrease the softening and denuding action. Pieces of clean domestic such as freshly laundered sheet or pillow case, cut the size and shape of the patches of eruption, are laid on heavy waxed paper and spread with a thick even layer of the salve, leaving no uncovered edges of the cloth as that tends to soak up the oily part of the salve and dry out the central part of the mixture. The paper is then cut the shape of the cloth, leaving about a quarter inch margin. This is turned over and applied to the corresponding areas of eruption and held in close apposition with a bandage. The paper, which is now over the salved cloth, prevents the salve from soaking into the bandage.

In spite of these directions the patient will often smear the salve on the affected parts and cover with a dry cloth and the wax paper. As this results in a more or less thin layer of salve which is absorbed by the cloth, the denudation is not accomplished.

Each time the dressing is changed the parts are wiped off with alcohol. As the effect progresses, the parts become more and more red and in areas of marked vesiculation, raw. The treatment of x-ray, soothing cream and mild salve is then repeated during another twenty-one day period.

We employ a similar method in reducing the thickening of chronic infiltrated eczema. The application in this condition consists of a modification of the old diachylon ointment made by rubbing equal parts of emplastrum plumbici and vaseline into a smooth paste with the aid of heat. The complete formula is:

Zinci oxidi 4
Pulv. amyli 8
Ung. picis liq. 4
Unguent. vaselini plumbici 32

The amount of tar can be increased according to the chronicity and infiltration to be contended with. This application is used during the twenty-one day intervals of x-ray exposures of one skin unit. The dressing is changed daily, the old application being wiped off with 1 per cent carbolized oil of sweet almonds. Application is kept on during a bath and removed immediately thereafter.

This paper should properly have been entitled "Meticulous Methods in Drudgery of Dermatology." We can console ourselves with the thought that genius has been defined as the infinite capacity to take pains, and that certainly the criterion of success in dermatological therapeusis is the willingness to give the time to take infinite pains.

In conclusion, I wish to suggest the revival of our round table discussions; to be devoted entirely to therapeutics. With no stenographic report, we could freely compare experiences and findings as to the efficacy of various commercial preparations for which so much is claimed, without the opportunity for the pharmaceutist to quote us in his advertising literature.

Every clinician has some methods, some hobbies, some gadgets, that would be helpful to his confreres. Every one has some subject on which he would like to get the opinion and experience of others. Personally, I should like to learn of the local efficacy of hot epsom salts solution, that panacea of the general practitioner, but never mentioned by the dermatologist. I wonder if others have shared my ups and downs with dermatitis venenata; the apparent immediate triumphs in some cases, and undoubted defeat in others.

What of the presence of anemia in eczema; what of its treatment with proteids and acids? What of the diet in acne; and so on. Finally, do not interpret my attitude as decrying the scientific phase of our work. The investigations and theories of our branch of medical knowledge make a most absorbing study, growing more fascinating as its complexity increases and its interrelation with other departments of medicine is developed. While it, too, includes much drudgery, the widening vistas of new ideas are a perpetual stimulation.

The therapeutic part is the daily grind, but it, too, has its compensations. Humanity does not seek us to help corroborate a favorite hypothesis, but to be relieved of dangerous and distressing conditions.

DISCUSSION (*Abstract*)

Dr. Emmett R. Hall, Memphis, Tenn.—Like Dr. Gaines, I believe that a more liberal discussion on treatment will be profitable to everyone. We realize that a proper diagnosis is all-important.

Successful treatment is an art, and the art cannot be acquired from textbooks alone. Experience is the most

valuable teacher. All of us have access to the same drugs, but we cannot use them with the same degree of skill. Little things and attention to details mean much. Dr. Gaines has called it drudgery in dermatology. I think it is an earnest desire to cure or relieve the patient.

I cannot go all the way with Dr. Gaines in his outline in treating acute tinea and squamous eczema. The principle is excellent; a wet dressing as he describes establishes free drainage, is antiseptic, causes an exfoliation of epithelial tissue, is cooling and will give relief when nothing else will.

Thiersch's solution seems to be entirely too mild, containing only 0.2 per cent salicylic acid and 1.2 per cent boric acid. By substituting for the water D. D. Hammamelis 250 c. c. he has added to Thiersch's solution about 4 per cent alcohol and I like the modification much better.

I think to change the wet dressing night and morning and by all means keep the dressing thoroughly wet is very important.

The use of wax paper excludes the air, prevents evaporation and increases the temperature. I have found that wet dressings are more comfortable where no material such as waxed paper, or oil of silk, that exclude the air and prevent evaporation, are used. True enough, it may keep some one busy keeping the dressings wet.

X-ray is a very valuable agent in treating tinea and eczema. There may be some difference of opinion as to frequency and size of dose.

Dr. Gaines (closing).—We are fully aware of the weakness of the solution and wished to call attention to its efficacy when applied continuously.

We have never had any untoward results from the use of the x-ray after these applications. On the contrary we have been criticised for being over conservative. We have been told by some of our confreres that we just played with the x-ray.

THE MASS CONTROL OF SYPHILIS*

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and
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Washington, District of Columbia

The term "mass control" is one which should have special appeal to every American physician and health officer. It implies efficiency. When used in connection with the control of syphilis the possibility of attaining success through the application of modern public health principles seems unlimited. In the control of any communicable disease, it is of fundamental importance to know when, where, why, and under what conditions the disease occurs. For syphilis most of these questions have been answered, yet many health officers, because of lack of appreciation of the seriousness of the problem, do not grasp these fundamentals. If we are to be successful in the mass control of syphilis through the application of modern methods, it is essential that the finer points in the prevalence of the disease be more generally known.

COMPARATIVE PREVALENCE OF SYPHILIS IN URBAN AND RURAL COMMUNITIES

During the past decade the U. S. Public Health Service and the American Social Hygiene Association, in cooperation with state and local health authorities, have attempted to define the scope of the venereal disease problem through cross section sampling of socially and geographically representative areas in the United States.[1] In these surveys, the number of individuals under observation and treatment for syphilis, as of a given date, have been reported by all sources of treatment within an area. The prevalence rates made available through these surveys are based on individuals seeking authorized medical care and do not account for the large number of individuals with syphilis who remain untreated, are self-treated or are treated by an unqualified or unlicensed practitioner. It is, therefore, necessary to make some reservations if the prevalence of syphilis based on these data in one type of community is compared with another.

Statistical information now available has been largely responsible for the present day conception that syphilis is primarily a disease most prevalent in urban communities. Further analysis, however, indicates that present day prevalence rates for syphilis may be distorted. Among factors which may be responsible for distortion are the higher "index of suspicion" on the part of the average urban physician and his greater willingness to treat syphilis. The urban physician is also materially assisted by the availability of efficient diagnostic procedures and the number of free or part-pay clinics attracts large numbers of patients from surrounding rural territory. On the other hand, a few infected individuals included in a large metropolitan area influence to a less extent the prevalence rate for the city than do the same number of infected individuals in a rural area. For example, in one of the areas surveyed by the Public Health Service data were assembled which revealed that 6 per cent of the patients seeking treatment in a metropolitan area

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resided in a rural community. This 6 per cent transferred to the contiguous rural area inflated the prevalence rate 49 per cent.

The prevalence rate for syphilis in cities of less than 50,000 population has been found to be a third higher than in large cities (7.4 and 5.5 per 1,000 population). In the report of the draftees examined during the World War, when the residence of the individual was definitely known, it was found that the relatively higher incidence of syphilis in the cities was due largely to its high incidence in the smaller cities. Four metropolitan areas studied, for example, had much lower rates for this disease than did even the rural districts. On an average the ratio of syphilis in rural to urban districts was 89 to 100, a much higher proportionate rate for rural areas than is generally accepted. Nelson,[2] in a very able analysis of present divergent opinion regarding the prevalence of syphilis and gonorrhea in rural and urban communities, states "that by the simple expedient of revising the reporting system nearly 200 of the 355 communities of Massachusetts, most of them small towns hitherto statistically free from syphilis and gonorrhea, became officially conscious for the first time of the existence of these infections." It is obvious, therefore, that treatment facilities must be developed in rural areas, or such areas will remain dependent upon the clinics and physicians in near-by cities through the use of modern transportation.

THE NEGRO IN RURAL AREAS OF THE SOUTH

The examination of collected data on syphilis in the United States reveals that in surveyed Southern localities, in which the percentage of Negro population is higher than that for the United States as a whole, the prevalence rate even in rural communities equals that for the country as a whole (*Table 1*). This highly syphilized Negro population in rural areas further complicates the treatment problem. The knowledge of this high rate of syphilis among Negroes led to a serologic survey of 33,234 Negroes in six counties in as many Southern states.[3] These rates show that 205 per thousand Negroes were infected with syphilis as evidenced by positive serologic blood tests (*Table 2*). A comparison of the results of such blood tests and the results of the one-day surveys of the Public Health Service indicates that only one out of every 10 Negroes infected with syphilis seeks authorized medical care. Further corroborative data on the lack of treatment for syphilis among Negroes is found in the report of Maxcy[4] that only 3 to 15 per cent of patients studied gave a history of ever having received antisyphilitic treatment. One of the writers, in conducting a study of untreated syphilis in the Negro in Alabama, serologically examined 2,299 adult male Negroes, of whom only 151, or 6.5 per cent, admitted ever having had modern treatment for syphilis. These data indicate the need for devising some practical and inexpensive method of treating these individuals.

Table 1

PREVALENCE RATES PER 1,000 POPULATION FOR EARLY AND LATE SYPHILIS IN SURVEYED SOUTHERN LOCALITIES IN WHICH THE PER CENT OF NEGRO POPULATION IS HIGHER THAN THAT FOR THE UNITED STATES

Locality	Total	Early	Late
Urban			
Lexington, Ky.	21.4	4.1	17.3
Dallas, Texas	8.9	3.9	5.0
Eldorado, Ark.	8.6	5.4	3.3
Jefferson County, Alabama	7.6	2.1	5.5
New Orleans, La.	5.8	2.3	3.6
Miami and Dade County, Fla.	4.9	1.6	3.2
Texarkana, Ark.	4.3	2.9	1.5
Paducah, Ky.	4.2	1.8	2.4
Total	7.3	2.5	4.8
Rural			
34 counties in Tennessee	4.4	1.7	2.7
16 counties in Mississippi	4.0	1.7	2.3
18 counties in Virginia	4.0	1.5	2.5
Scott County, Kentucky	3.3	2.5	.8
Total	4.2	1.6	2.6
Total urban and rural	5.1	1.9	3.2
Total United States	4.3	1.2	3.0

F.—10/15/36

Table 2

THE PREVALENCE OF SYPHILIS IN THE NEGRO BASED UPON SEROLOGIC EXAMINATION OF UNSELECTED INDIVIDUALS IN SOUTHERN RURAL AREAS

Locality	Grand Total Examined	Number Positive	Rate per Thousand
Macon County, Alabama	3,684	1,474	398
Glynn County, Georgia	7,408	1,933	261
Bolivar County, Mississippi	3,693	869	235
Pitt County, North Carolina	11,931	1,494	125
Tipton County, Tennessee	2,706	691	255
Albemarle County, Virginia	3,812	339	89
Total	33,234	6,800	205

AGE AT THE TIME OF INFECTION

Another important point in the mass control of syphilis is a determination of the age group in which the peak of the infection occurs. We are prone to think of syphilis as a disease of young adults, rarely thinking of the high infection rate among individuals in their teens. True, the mean age is from 20 to 28 years, but of 500,000 fresh infections each year, 100,000 occur in individuals between the ages of 11 and 20! In fact, annually there are 11,000 fresh infections among children 11 to 15 years of age (as shown in *Table 3* and *Fig. 1*). Approximately 80 per cent of the infected individuals acquire syphilis before middle age. Whatever is to be done in the way of control, therefore, must be so planned that it will provide early and adequate treatment for this group of individuals who are economically in the years of lowest earning power. This appallingly high rate among young individuals demands action.

THE PATIENT PREFERS THE CARE OF THE PRIVATE PHYSICIAN

Since two-thirds of the syphilitic patients under treatment in the United States are under the care of private physicians it is obvious that the patient prefers treatment for syphilis from this source to that of public or part-pay clinics. Yet only 20 per cent of the population can pay for adequate care of syphilis at private rates.[5] This tendency on the part of the patient to seek treatment for syphilis from the private physician, whether it can be paid for or not, places an unfair burden on the physician. It relieves the health officer of a proper but onerous financial responsibility. Because the private physician cannot indefinitely continue to treat a patient and provide expensive drugs it encourages lapses and consequent development of communicable relapse.

THE APPLICATION OF MASS CONTROL

Everyone must appreciate that the mass control of syphilis does not connote the blind administration of one of the arsphenamines and heavy metals to any and every patient who is admitted to a clinic. The early syphilitic patient is the ideal candidate for mass control, since a planned schedule of treatment may be adopted and adhered to with these patients. Treatment by the continuous alternating method, as advocated by the Cooperative Clinical Group,[6] should be administered only in early syphilis. A slightly modified form may be employed in early latent syphilis when the clinician is certain that no contraindications for such treatment exist. In late syphilis each patient should be given individual consideration. The demonstrated effectiveness of these treatment schemes in preventing muco-

Table 3

AGE OF INDIVIDUAL AT TIME OF ACQUIRING SYPHILIS

(Estimate based on age of 3,244 cases of early syphilis and the annual incidence of 500,000 fresh infections of syphilis in the United States)

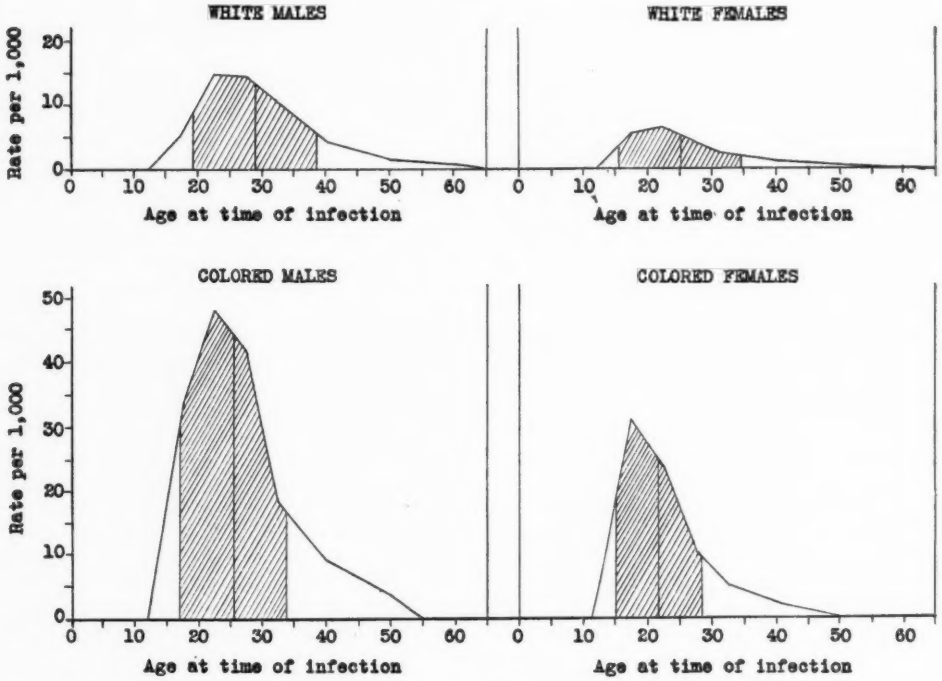
Age at Time of Infection	White				Colored			
	Male		Female		Male		Female	
	Cases	Rate per 1,000	Cases	Rate per 1,000	Cases	Rate per 1,000	Cases	Rate per 1,000
11 to 15	1,406	.26	4,682	.88	1,778	2.85	3,566	5.68
16 to 20	28,399	5.50	29,652	5.77	20,026	33.62	20,244	30.91
21 to 25	71,702	14.99	32,173	6.59	26,670	48.17	15,416	23.73
26 to 30	62,985	14.45	19,088	4.34	20,774	41.50	6,035	10.56
31 to 35	44,989	10.85	10,204	2.48	7,767	18.63	2,358	5.27
36 to 45	34,867	4.33	8,883	1.17	7,112	9.24	2,140	2.65
46 to 55	11,529	1.86	3,481	.61	2,339	3.89	165	.31
56 to 65	3,374	.83	960	.25	.....	.....	.....	.....
Over 65	562	.18	360	.12	.....	.....	.....	.....
Mean		28.91		25.15		25.42		21.68
Mode		24.45		22.96		23.55		20.06
Standard deviation		9.69		9.56		8.36		6.68

GRT—9/22/36

Fig. 1

AGE OF INDIVIDUAL AT TIME OF ACQUIRING SYPHILIS

Estimate based on age of 3,244 patients with early syphilis and the annual incidence of 500,000 fresh infections of syphilis in the United States.



cutaneous and clinical relapse provides our greatest hope for controlling syphilis.

A second strategic point of attack lies in the possibility of the prevention of the prenatal transmission of syphilis from the syphilitic pregnant woman to her child. The fact that this prevention may be accomplished by adequate treatment in more than nine cases out of ten is so well known that the statement needs no further comment. Patients with early acquired syphilis and pregnant women with this disease must be found early and kept under treatment. The medical follow-up of such patients to insure the administration of adequate treatment is also a fundamental point in mass control.

POSSIBILITIES OF EPIDEMIOLOGIC METHODS IN MASS CONTROL

No discussion of mass control would be complete without some slight consideration of present day epidemiologic methods. The time is rapidly approaching when the efficiency of any clinic will be gauged by the effectiveness of its case finding. The same statement applies to the individual physician. Experience in the tracing of sources of infection and contacts indicates that frequently from twelve to fifteen or more individuals infected with early syphilis may be discovered for whom treatment would otherwise be delayed or never administered had such epidemiologic principles not been applied. Through the early diagnosis of and the application of treatment to such cases mass control is further facilitated. Such methods offer a most subtle maneuver, which has as its purpose the severing of the ramifying links in the chain of syphilis transmission.

NEED FOR THE DEVELOPMENT OF TREATMENT FACILITIES

What are the needs of the country with regard to the development of facilities for treatment? The Public Health Service[1] has estimated that there are more than half a million new cases of

early syphilis seeking treatment each year. The provision of adequate treatment for all patients with syphilis should be the ultimate goal of every health officer. Adequate treatment in early syphilis is generally accepted as consisting of at least thirty injections of one of the arsphenamines and accompanying heavy metal. Consider the extent of this treatment problem. For the proper treatment of patients with early syphilis only, the administration of 15,000,000 doses of one of the arsphenamines and 15,000,000 doses of one of the heavy metals would be required annually. A simple mathematical average reveals the fact that the administration of such an amount of treatment would require 100 injections annually of one of the arsphenamines and one of the heavy metals by every physician in the country. Yet in the prevalence studies just referred to it has been established that while private physicians treat 64 per cent of all patients, 5 per cent of the physicians treat more than half of the private patients. In other words, 7,500 of the country's physicians would be administering 9,000,000 injections each of the arsphenamines and heavy metals. Syphilology would truly be a profitable specialty, if the patient could pay and the doctor collect!

But what percentage of patients now receive 30 doses each of one of the arsphenamines and one of the heavy metals? Experience indicates that in some of the better type clinics throughout the country adequate treatment of this type is received by only from 15 to 25 per cent of the patients with early syphilis. As far as is known, no data exist which indicate the extent to which the physician in private practice administers adequate treatment to his early syphilitic patients. Some indication of the relative inadequacy of present day treatment is gained by a study of *Table 4*. This table presents the estimated number of doses of the arsenical drugs based on the amount in grams manufactured by the various commercial laboratories in the United States. It has already been pointed out that 15,000,000 doses of the arsphenamines are required if adequate treatment is to be given to all of the patients with early syphilis who seek it each year. A study of the accompaying table, however, indicates that only between 4,000,000 and 5,000,000 doses of all of the various arsphenamines are manufactured in this country. When it is appreciated that this limited supply is used not only in the treatment of early syphilis but in the treatment of patients in the latent and late stages of this disease, as well as of patients with other diseases, the relative inadequacy of treatment in early syphilis may be better appreciated.

Table 4

ESTIMATED NUMBER OF DOSES OF ARSENICAL DRUGS SOLD IN THE UNITED STATES IN 1933, 1934, AND 1935

Kind of Arsenical Drug	Number of Doses Sold* 1933	1934	1935
Old arsphenamine	244,300	299,900	309,000
Neoarsphenamine	3,892,800	4,313,000	3,795,600
Sulpharsphenamine	246,500	350,000	185,000
Silverarsphenamine	16,000	24,100	21,000
Neosilverarsphenamine	1,000	1,400	900
Total doses sold	4,400,600	4,988,400	4,311,500

*Estimate based on 0.4 gm. average dose for old arsphenamine and 0.6 gm. average dose for the other drugs.

If the patient cannot amply compensate the private physician, should the latter be expected to carry the whole burden of control? Should we not indicate a method through which results can be obtained in a more efficient and economical way? Is that way not the clinic, whether it be affiliated with a hospital in an urban community or a traveling unit for the management of syphilis in the rural South? If the physician could be relieved of the non-paying patients with syphilis amenable to mass control he would be free not only to treat adequately the well-to-do patients, but also to lend his assistance in the well organized control program for the masses. Whatever may be the method of choice, the assistance of the private physician is essential to the success of any syphilis control program. It is he who is usually consulted by the patient with early syphilis, he who should make the diagnosis. His is the first interview with the patient, an interview which should be the dominating influence on the patient's future behavior.

The following, then, may be said to be the fundamental points in the mass control of syphilis:

(1) Experience indicates that in densely populated areas the prevalence rates are lower than in smaller cities. This may be due to the more limited development of treatment facilities in the smaller cities.

(2) Serologic surveys in Southern rural areas show that about one out of every four Negroes is infected, but not more than one Negro in ten with syphilis seeks authorized medical care. Adequate treatment should be provided for these individuals.

(3) Annually, one hundred thousand adoles-

cents acquire syphilis before they develop earning capacity.

(4) That present treatment facilities are inadequate is evidenced by the fact that the total amount of all of the arsphenamines sold in the country annually is sufficient only for the proper treatment of one-third of the patients with early syphilis.

(5) The tremendous financial burden of providing treatment for syphilis today is borne by the private physician. This burden should be shared with the health officer.

(6) Mass control is most applicable to the patient with early syphilis who can be placed on a planned treatment schedule.

(7) Discovery of contacts and return of delinquents to treatment are strategic points in the mass control of syphilis.

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DISCUSSION (*Abstract*)

Dr. Dudley C. Smith, University, Va.—"Mass control" of syphilis includes "mass detection or diagnosis" and "mass treatment." Detection of cases of syphilis depends on the clinical ability of physicians, routine laboratory tests and investigation of all contacts of syphilitic patients.

In the early stages of syphilis, tracing source cases and examining associates is probably the most effective way of getting infectious cases under treatment promptly. Clinical acumen and serological tests are more important in recognizing syphilis in its later stages. Widespread use of serological tests detects also a number of early infectious syphilis. In a recent survey of the employes of a small hotel two cases of secondary syphilis were found. In my opinion greater efforts to stimulate serological tests on all groups of the population, those seeking medical advice as well as apparently healthy individuals, should be made. These tests as a part of routine health examinations for everybody would in a few years locate many foci of infections.

We have seen recently a great increase in interest to detect and treat early syphilis in order to prevent further spread of the disease. There is another preventive medicine aspect of syphilis which is equally worth promoting. The discovery and proper treatment of syphilis in the early latent period prevents in a great majority of cases serious later visceral damage to the cardiovascular apparatus, nervous system, etc. In my opinion, this is preventive medicine of the highest order.

Dr. Vonderlehr suggested two lines of attack: (1) the increased use of the whole medical profession, and (2) the establishment of more clinic facilities for the indigent syphilitic. Both of these general measures should be used to the limit and each coordinated with the other. It will take all forces marching at double quick time to catch up with and defeat the enemy "Syphilis."

The cost of a campaign against syphilis has been overemphasized. When the cost of diagnosing and treating syphilis is compared with the expense involved in most other infectious diseases it is found to be relatively smaller. At the present day prices 40 doses of an arsenical, 60 doses of a bismuth preparation, and a pound of mercurial ointment (these being sufficient to cure the average case) can be purchased for less than the cost of one week's hospitalization of a tuberculosis patient in a public sanatorium. This, of course, is no reason for relaxing the campaign against tuberculosis, but it is a fact which makes it possible to start a much more vigorous campaign against "The Great Imitator."

MORBID ANATOMIC CHANGES FOLLOWING ARTIFICIAL FEVER, WITH REPORT OF AUTOPSIES*

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Of therapeutic agents, heat in various forms is probably one of the oldest. However, its most widely used and oldest form has been that of a local application. During the last quarter of the Nineteenth Century an English physician, Dr. Buisson,[1] under the impression that he had hydrophobia, took a steam bath "and at 42 degrees (127 Fahrenheit,") he says, "I was cured." His, as we now know, was an ordinary case of imaginary rabies. Nevertheless, the Buisson bath was adopted by many antivivisection societies in England as an antidote for hydrophobia. Probably the first planned attempt to use fever or increased body heat as a therapeutic agent was made by Wagner von Jauregg in 1918 in the malarial treatment of paresis. Since then malaria and other biological substances have been used and, in addition to these, various mechanical devices have been constructed.

It would seem, however, that the therapeutic value of artificial fever, induced by whatever

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means, has been stressed and the physiological group of phenomena following this type of therapy has been more exhaustively studied than the pathologic changes which frequently occur, occasionally with fatal results.

The Council on Physical Therapy of the American Medical Association in 1935 reported the treatment of 4,809 patients with 29 deaths, a mortality of 0.6 per cent. Hench[2] reports only one death in over 300 cases. Desjardins[3] reports one fatality in 362 patients who received 1,810 sessions of treatment, or less than 0.3 per cent. It should not be lost sight of then, that occasional deaths do occur following artificially induced fever.

The efficacy of heat as a therapeutic agent for dealing with certain diseases cannot be questioned. The two diseases which respond best to fever therapy are syphilis and gonorrhea. Hench[2] reports in 143 cases of gonorrheal arthritis 100 cases received complete relief and 14 received marked relief. In 139 cases of gonorrhea limited to the genito-urinary tract, 118 obtained excellent clinical and bacteriologic results. Gonorrheal involvement of the eye, as shown by Hasler and Spekter,[4] responded well. The results obtained in the treatment of syphilis have been well covered in the literature.

The purpose of this paper is to record and discuss the postmortem findings of two of our own cases and four collected from the literature on the subject. A case of sunstroke is also presented for comparative purposes. In no way is it an attempt to detract from the value of this form of therapy, but to gain a better understanding of the physical alterations produced in the body by sustained high temperature.

Schnabel and Fetter[5] report a fatality resulting in treating a 12-year-old child with chorea but without heart disease. Cyanosis and shock appeared one hour and a half after onset of the treatment. Stopping the treatment and treating for shock resulted in some improvement, but death occurred nine hours later.

At autopsy, there was edema of the brain, acute passive congestion of the brain and viscera, mild parenchymal degeneration of the adrenals, and slight hyperplasia of the thymus.

Watts[6] reports a case of multiple sclerosis that after 4 or 5 hyperpyrexia treatments had a vascular collapse and lived thirty-six hours. The autopsy findings were: focal necrosis in the liver; brain edema; cerebral and pulmonary congestion; thrombosis of the lateral sinuses, and no evidence of necrosis or degeneration in the adrenals.

Hartman[6] discusses two cases that came to autopsy and his conclusions are: the pathologic changes in the human cases and experimental animals are engorgement of the blood vessels, especially the capillaries, hemorrhage and degeneration. The most vital are hemorrhagic changes in the brain, a hemorrhagic pneumonia and degenerative changes in the adrenal cortex.

The method of producing hyperpyrexia used in the following cases was radiant heat produced by electric light bulbs. The patient was protected by forcing sodium chloride and fluids the preceding day, and during the treatment the patient was under constant supervision; his blood pressure being taken every 15 or 20 minutes.

Case 1.—A white male, aged 23, entered the hospital complaining of a burning sensation in the penis for the previous ten days and an urethral discharge for three days. The urethral discharge was creamy and inguinal and postcervical lymph nodes were enlarged. His hemoglobin was 14.4 gm.; white blood count was 4,100. The urethral smear showed many Gram-negative, intracellular diplococci.

Three days after admission, hyperpyrexia treatment was started. After two and a half hours he went into shock and had a convulsion and following this became stuporous. Treatment for shock resulted in a slow response and a few hours later electrocardiogram showed a slight abnormality suggesting myocardial damage. At this time the urine had a few granular casts and a four-plus benzidine reaction. The next day improvement was moderate, but he complained of headache and "gas on the stomach." On the morning of the third day following the treatment, after vomiting once, he complained of anesthesia of the left leg. Examination revealed: anesthesia and paralysis of both lower extremities, bilateral papilledema, bilateral subconjunctival hemorrhage, imperceptible pulse, stertorous breathing and weakness of both upper extremities. A lumbar puncture yielded a turbid fluid containing numerous red cells in the centrifuged specimen. The patient died thirty-five minutes after his initial symptom, the vomiting. Autopsy was performed seven and a half hours after death. Both lungs were congested and there were submucosal hemorrhages in the trachea. The liver weighed 2,350 grams and showed diffuse early necrosis. The spleen weighed 380 grams and was very

CASE I

CHART SHOWING BLOOD PRESSURE, TEMPERATURE, PULSE AND RESPIRATION OF TWO CASES TREATED BY HYPERPYREXIA WITH FATAL RESULTS

	B.P.	T.	P.	R.
Admission	150/90	36.7	80	20
Start treatment	140/90	36.7	84	20
End treatment	90/40	41.5	160	78
12 hours				
24 hours	130/90	37	80	20
48 hours	130/90	37	78	20
72 hours		37.4	70	20
		Death		

soft. Each kidney was swollen and weighed 275 grams. There were numerous petechial hemorrhages in the pelves. The largest of the hemorrhages was 1 cm. in diameter. The epithelial cells of the convoluted tubules had early necrotic changes and the capillaries were engorged with red cells. There were subendocardial and myocardial petechial hemorrhages. Some of the muscle fibers of the heart and diaphragm were hyalinized. All of the viscera were congested.

Case 2.—This patient, a white male, aged 28, was admitted with an urethral discharge of seven weeks duration and hematuria of two and one-half weeks duration. According to his past history, every hot summer he had three or four heat strokes which made him unconscious for about three hours. During hot weather he frequently experienced tinnitus, scotomata and vertigo. He contracted syphilis in December, 1933, and early in January of 1934 his Wassermann reaction was 4 plus. He had about 150 treatments and in November, 1935, the Wassermann reaction was negative. He contracted gonorrhea in July, 1934; he had been treated for chancroid, and buboes.

The present illness began with an urethral discharge seven and a half weeks before admission. Three days later he noticed pain in the back and dysuria, particularly at the end of urination. Five weeks later he began to have hematuria.

The physical examination showed an old scar on the prepuce. There was pain on percussion over the bladder; the prostate and seminal vesicles were moderately enlarged and tender. The hemoglobin was 14.4 grams and the white blood count 9,000. The urine showed many white cells and a three plus benzidine reaction. The urethral smear had many Gram-negative biscuit-shaped intracellular diplococci. The Kahn reaction was three plus.

His hyperpyrexial treatment was started three days after admission. After one hour of a temperature of 41.5° C., his temperature suddenly rose to 42° C., he went into shock, began to have generalized convulsions and became stuporous. He was immediately removed from the box and treated for shock. He improved slightly for the next 16 hours, after which his temperature began to rise, the convulsions began again, and he died seven hours later.

The autopsy was performed three hours after death. There were petechial subendocardial and myocardial hemorrhages. The lungs were congested and many of the alveoli were filled with red cells. The liver weighed 2,300 grams and had diffuse early necrotic changes. Each kidney weighed 350 grams, the epithelial cells had early necrotic changes and the capillaries were engorged. The spleen weighed 450 grams and was very soft. Sections of the brain showed the astrocytes to be swollen and beginning to break up.

CASE II

	B.P.	T.	P.	R.
Admission	106/56	36.5	88	20
Start treatment	120/70	36.8	84	18
End treatment	72/15	42.5	170	24
12 hours	100/70	38.8	118	26
24 hours	0	41.9	?	44
		Death		

*Case 3.**—This case of sunstroke occurred in a white male, aged 33. He was admitted to the hospital with a history of exposure to the sun, followed by collapse and unconsciousness. His temperature was at least 110° F. (that was the highest the thermometer would record). His urine had three plus albumin and one plus granular casts. He lived three days and his temperature remained above 103° F. He continued to be stuporous and a few hours before death he became jaundiced.

The autopsy showed early necrotic changes in the liver and epithelial cells of the kidney tubules. There was also congestion and extravasation of red cells in the kidneys and lungs.

DISCUSSION

To dissipate excessive heat the body utilizes three main mechanisms. One is perspiration with resultant evaporation. Its effectiveness is the result of changing water from a fluid to a vapor at body temperature. This requires 5.82 times the heat required to raise water from freezing to boiling.[7] Heat is also dissipated by increased respiration expelling hot humid air in an attempt to increase evaporation.

According to Desjardins[3] the effects of fever are: (1) increased flow of blood; (2) increased action of the sweat glands; (3) increased rate of respiration; (4) increase in metabolism (about 8 per cent for each degree rise in temperature); (5) increase in the number of leukocytes; (6) decrease in the sedimentation rate. (7) In an environment warmer than the body the cutaneous capillaries become dilated as a response to stimuli from the central nervous system. (8) This dilatation aids in a more rapid flow and a greater surface area exposure of the blood.

One of the results of these reactions is a depletion of fluids and chlorides due to excessive perspiration. Another is alkalosis due to hyperventilation lowering the carbon dioxide tension. The dilatation of the capillaries increases the capacity of the vascular bed and the depletion of fluid lowers the blood volume.

The clinical course and pathologic findings of these cases are in accord with the accepted body reactions to increased temperature. Although the fluid loss can be partially replaced, the second case had a hematocrit of 56.2 per cent.

The second case did not have an alkalosis two hours after the treatment, for his carbon dioxide combining power was 48 volumes per cent. However, this may be due to the fact that

*Permission to use this case was given by Dr. McMicken Hanchett, of Council Bluffs, Iowa, and the autopsy report was obtained from the Pathology Department of the Jennie Edmundson Memorial Hospital of that city.

his respiration was never over 28. In the first case, however, the respirations at one time were 78.

Two factors may account for the hemorrhages. The hemorrhage may be due to the extreme capillary dilatation stretching the walls and allowing the red cells to extravasate. The other factor is that there may have been actual capillary damage. This was not demonstrated anatomically.

The explanation of shock may rest on the central nervous system control of the caliber of the blood vessels. The dilatation of the capillaries, both cutaneous and visceral, would increase the capacity of the vascular bed. To a certain extent this is normal, but if the temperature was so great that the vasoconstrictor center ceased to function, the resultant dilatation combined with the fluid loss would create such a disproportion between volume and capacity that shock would result.

The liver damage may be due directly to the heat or to intoxication. This intoxication may result from the increased metabolism piling up katabolic products so rapidly that the liver is overwhelmed.

The case of sunstroke compares closely with the cases of artificial hyperpyrexia.

There is a definite variation in the individual susceptibility to heat. This is shown by our two cases. One had a history of heat susceptibility and had a shorter, more violent course than the other.

SUMMARY

Two cases of death following artificial hyperpyrexia and one of sunstroke have been presented. In addition, four cases in the literature have been reviewed. The most consistent findings in these cases were congestion, petechial hemorrhages, and diffuse early hepatic necrosis. Evidence of damage to the central nervous system was found in only one case. This was characterized by degenerative changes in the astrocytes.

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DISCUSSION (*Abstract*)

Dr. Walter M. Simpson, Dayton, Ohio.—During the past five years we have subjected 590 patients to approximately 20,000 hours of artificial fever therapy, in the Department of Fever Therapy Research at the Miami Valley Hospital. With the exception of a recent patient with fulminating meningovascular syphilis and rapidly progressive blindness, who was treated with full knowledge of the grave risk involved, and who died 36 hours after a single short treatment, no deaths have occurred which could be attributed to the artificial fever treatments. All of our patients have been subjected to a thorough diagnostic survey by the physicians in the Department of Fever Therapy Research to determine eligibility for fever therapy. Special studies are made of the cardiac, vascular and renal functions, including electrocardiographic studies, basal blood-pressure determinations, renal function tests and blood chemical analyses. It is our practice to determine the individual patient's tolerance for artificial fever therapy by giving a short trial treatment before undertaking a course of treatments requiring higher temperature levels over a longer period. Careful inquiry is made as to a history of heat stroke, sun stroke or a tendency to sunburn readily. If such a history is obtained the patient is either treated with exceptional caution or denied treatment.

This report again emphasizes the fact that artificial fever therapy by physical means is not a simple undertaking. In the hands of unskilled or unscrupulous persons it is fraught with danger. Even in the hands of skilled workers, as in the present instance, occasional accidents may occur. History is repeating itself in the attempts of certain manufacturers to exploit the field by utilizing high-pressure sales methods. Some of the apparatus now available through commercial sources is inadequate and dangerous, and is sold to any physician without thought of adequate training of the supervising physician and his technical assistants. Three important considerations are involved in the proper application of artificial fever therapy. The first is the thorough training of the supervising physician in the physical and the physiologic aspects before the work is undertaken. The second is the adequate training of the nurse-technicians; exceptional alertness and intelligence are primary qualifications for nurse-technicians who engage in this work. Training in ordinary physiotherapeutic procedures does not qualify one for work in this field. The third, and perhaps the least important consideration, is the apparatus. The undertaking is in many respects comparable to a major surgical

operation, particularly as regards the necessity for a careful diagnostic survey to determine eligibility and the constant attention to the patient by physician and nurse-technician during the long treatment. It is well to re-emphasize the obvious but too often disregarded fact that skill of personnel far transcends in value the perfection of material.

It seems quite apparent that artificial fever therapy by physical means should be restricted to institutions in which the physician and nurse personnel have received adequate preliminary training. Moreover, the likelihood of success in this work is greatly enhanced if it is done by physicians and nurses who devote full time to it. To give only occasional treatments in a haphazard manner is to invite disaster. The production of effectual artificial fever is not adaptable to ordinary office practice. Unless these precautions are exercised this important adventure in therapeutics is almost certainly doomed to a period of discredit not unlike that which followed the introduction of roentgen rays. In the hands of skilled and devoted workers this form of therapy seems destined to occupy an increasingly important place in the therapeutics of several diseases which have not yielded to other forms of treatment.

Dr. Harry C. Schmeisser, Memphis, Tenn.—I recently saw a case in which the congenital absence of sweat glands was the cause of a continuous spontaneous hyperpyrexia. Such an individual, while otherwise healthy, might respond poorly to fever therapy.

Dr. Wilbur (closing).—Since the reaction of an individual to heat is not absolutely predictable utmost caution needs to be observed in handling these patients. A history of susceptibility to heat should be considered at present a contraindication to this form of therapy. That there is a definite risk in this form of therapy is proven by the previously quoted mortality of 0.6 per cent. Our mortality rate has not exceeded 0.4 per cent in at least 500 treatments. In this number of treatments the two fatalities comprise two definite types of cases. One is a death that can be explained by the past history of heat stroke. The other is an unexplained death. It occurred in a 23-year-old white male who showed no contraindication to hyperpyrexial therapy.

We believe that as this form of treatment is more extensively observed there will be a definite small percentage of unexplained and unpredictable fatalities. Such an observation would not be surprising because many forms of therapy have a certain death rate. Commonly used anesthetics are sometimes responsible for death and deaths have been reported as due to such a usually safe drug as acetylsalicylic acid.

The maintaining of these fatalities at a minimum is probably dependent on the careful preparation and supervision of these patients. Further understanding of the problem will be gained by careful and complete autopsies of such fatalities as do occur. The fact that some of the fatalities may be due to an allergic response to heat should be more thoroughly investigated and testing patients for this sensitivity may be of value. This testing is used in this institution in that the first treatment is always started as a test and the conduct of the patient is governed accordingly.

A PSYCHIATRIST'S VIEW OF THE PROBLEM OF PREVENTION IN PEDIATRICS*

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At the meeting of the American Academy of Pediatrics last June, Dr. H. K. Tenney‡ raised the question as to whether we could (and should) prevent a great number of the difficulties which we were discussing through the development in parents of what he ingeniously termed "the third child attitude." With this view there was general agreement. An interesting and quite convincing effort along this line is Dr. Tenney's charming little book, "Let's Talk About Your Baby." These disarming, straightforward discussions appeal as being of real help, even though the psychiatrist sees a deeper set of problems involved in the development of this attitude. It is these last difficulties which I wish to discuss today. My plan is much more one of talking over with you certain problems than of answering the questions which they raise. On this basis we review five of the ordinary, usual problems which appear in all family relationships, assaying in this way what seem to be five definite hazards in the development of this "third child attitude."

First of all, there is this matter of having children. A large number of families look to the child to answer certain fundamental questions that have been raised by earlier personal problems. From the time that we are adolescent we feel that we are going to make up for the mistakes of our own lives through our children. The pediatrician frequently fails to realize the terrific burden that is placed on the child through these needs of the parents. When we know that a child is coming we "hope that it will be a boy" or "hope that it will be a girl," not having the courtesy to await its arrival before we decide what role we want it to fill. Before it is a month old we see that he smiles like Uncle Ned (who is in the penitentiary) or has a forehead like Aunt Jane (a great feminist leader). Somehow or other (through, perhaps, the teachings of the pediatrician or the general medical man), parents must realize that it is unfair to

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the child to have him come into the world until they do not *need* to have him come into the world. The demand for "the third child attitude" is just grotesque mockery as long as the child has to carry the load of broken hopes and unfulfilled dreams. We have made considerable progress in the problems of weaning children from their parents. I think that we have done little or nothing in the equally important matter of weaning parents from their children. And we would be blind if we did not realize that this dependency of the parent upon the child goes back many years to base its demands upon the most poignant tragedies and deepest longings of the parents' lives. The answer (and I feel that this is a distinctly medical problem if we are really interested in prevention) lies in the development of internal resources, in the development within ourselves of the answers to our ventures in living. It all has to do with building one's house by all means among many other houses, for there lies enrichment of life, but in building it so that if all the other houses were to fall down, it would still stand.

The second problem is that of the distortion that comes from love. The pediatrician asks for a certain objectivity, and certainly for the problems of weight and diet and sleeping it is better that the parent have that same rather detached view of the situation that the doctor or nurse or teacher can have. But love which gives security and which solidifies the family and which is the most important thing which the family gives to the child, is a two-edged sword that makes us care, makes us worry, makes us see the lives of those whom we love in an absolutely different way from the lives of those whom we do not love. We can always easily solve our neighbor's problems. But they are just as easily solving ours! The kindergarten teacher who does a marvelous habit-training job with other peoples' children finds this a very different proposition when she marries and has her own. The demand of the modern pediatrician and psychiatrist for objectivity, for cool assay of the importance or non-importance of the elements of the problem of growing up, may succeed in accomplishing the little things of life but only at the cost of the one really worth while thing which parents give to their children.

The child represents to a very large extent the wife's contribution to the family. Admittedly, in days of growing economic importance of women, this contribution may lose in its importance, but in most of the families in which we are working the wife still thinks of the production and rearing of children as her peculiar part in the venture. Indeed this will, at the least, remain the one thing she can do which the husband can not do. The worry, the balancing of little points, the magnifying of details, that urging insidious sort of doubt that everything will come out all right, all those things that are such enemies of the third child attitude, all those things are the attendants upon anything that is really important to us. When you say to the mother, "Oh, don't bother about Johnny's weight, or Johnny's spinach," you are really saying to her, "After all, this matter of Johnny's growing up is not important." I am quite ready to admit that this ought not to be so, but am just as ready to claim that it is so.

The fourth of the difficulties is the most subtle and might be considered the most important because it is hardest to attack. I refer to the problem of disentangling the threads of love and dependence. It is easy to think that those who depend upon us love us. And the other side of the picture is true, too, that it is very difficult to love those who do not need us. You see this problem most clearly when the children are adolescent, when both they and the parents are rather boldly presented with the question of the continuance of a love relationship when it is so necessary that the children no longer depend upon their elders. After all, as we grow up perhaps the most complicated of our problems is that of clearly seeing that we continue to love our parents as we more and more become independent of them. For the baby you must not forget that much of the smothering solicitousness, much of the adding up of small details of care, much of what works so sincerely against the third child attitude, comprises the very necessary symbol of the existence and strength of the love tie.

Finally, there is the queer fact that life is never really a matter of the solution of problems of relationship, but rather of adjustment to the fact that we have these problems. This goes hard with the medical man who likes to think that he "cures" people. (However, he is learning even in the physical field that his task is, through regimen, to get persons to live comfortably with certain physical states rather than to cure those states in the usual sense of this word "cure.") In the field of personal relationships the whole matter of how much love or how much punishment or how much jealousy or

how much charity, how much objectivity, how much worry, the matter of all these drives is a changing set of constellations which never has any final answer. Patients come to us looking for absolute statements. They want to know what is safe and what is right. We have to develop the point of view that nothing is completely safe and nothing is completely right. Such and such if done today will produce certain results. As long as we live we have problems, ways by which we are trying to get some answer out of life. These problems are never solved. There is never a time that one can say, "Well, that's off my mind." The question is that of adjusting people to the fact that they will always have these problems rather than of leading them on (as we doctors have) to the belief that we solve or do away with problems.

This is not a pessimistic talk. Pessimism lies rather in that happy, naive attitude which refuses to see the difficulties that lie in the road of our progress. I have not meant to say "the third child attitude" is impossible, because actually parents do develop a third child attitude. I have meant only to bring to your attention certain fundamental problems involved in the development of this attitude and to indicate that it is at the level of finding some answer to them that the pediatrician's program of prevention must begin. As we understand the situation better, I should not be surprised if we found the following:

(1) That "the third child attitude" to a considerable extent represents the development in parents' lives of a belief that they have found one or another answer to these problems. This is not to say that the answers have been wise, but that they have at least removed the problem from the realm of confusion.

(2) That "the third child attitude" is not entirely a desirable asset. Even admitting that it relieves the pediatrician of many bothersome visits and the child of many over-solicitous urgings, may it not also represent a certain cessation in family growth, in family adventuring, in the sort of change that is life? At least until we have data to show (and the data we now have are decidedly to the contrary) that the third child is any better adjusted, any less vulnerable to the various pitfalls in the problem of life, than are his predecessors, the problem of prevention in pediatrics, at least so far as the psychiatrist sees it, remains an exceedingly nebulous affair.

MATERNAL MORTALITY IN GEORGIA DURING THE YEAR 1935*

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It has been long recognized that the maternal mortality rates in the Southern states have been higher than those in other parts of the United States. It has been shown that the maternal mortality rate in this country is 6.4 to 6.7 per 1,000 live births, while in Sweden and Italy it is 2.6 to 2.7 for the same number of live births. These figures represent the highest and lowest rates. It is also a fact that no appreciable improvement in our own rates has been made for several years. Among obstetricians, particularly, there has been systematic inquiry and serious thought regarding the causes for this situation. The White House Conference Studies on Prenatal and Maternal Care and Child Health and Protection created much interest. It showed that in the United States' birth registration area there are annually 15,000 maternal deaths, 86,000 stillborn infants, and about 80,000 more dying in the first two weeks of life.

Before the number of maternal mortalities per year can be reduced, it is necessary that we understand the causes of our increased rate. A campaign of investigation should proceed through agencies of the medical profession, particularly those of public health. Believing that the following is an example of the problem faced in many other Southern states and may serve as a stimulus for similar surveys, the author appreciates the opportunity of presenting this study on maternal mortalities in Georgia during 1935.

The material from which this paper was prepared is from the report of the Committee for the Study of Maternal Mortality to the House of Delegates of the Medical Association of Georgia, Savannah, April 23, 1936. The author was chairman of this committee.

METHOD OF SURVEY

A copy was made of the certificates of puerperal deaths as reported to the Georgia Bureau of Vital Statistics. These copies made on the tenth day of each month included the certificates of the previous month. Therefore, each

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death was investigated within forty days after it had occurred. A questionnaire, accompanied by a return addressed and stamped envelope, was mailed to the attending physician or midwife. In a few instances it was necessary to write several letters before the desired information was returned. Some physicians totally ignored repeated communications. However, as a rule, the attendant had a vivid recollection of the case and was cooperative with the committee.

ACKNOWLEDGMENT OF ASSISTANCE

The staff of the Bureau of Vital Statistics, under the supervision of Dr. T. F. Abercrombie, rendered prompt and efficient service to the committee. With its cooperation it was possible to accumulate the desired data within a few weeks after a death had occurred. Previous experience has shown that it is more difficult to obtain the information after a lapse of several months or a year.

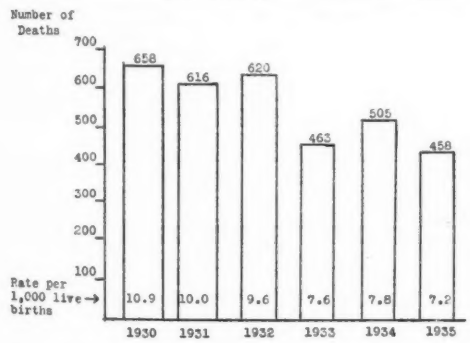
The hearty cooperation of the physicians, midwives and hospitals of the State made it possible to complete the survey. Without their aid and support it would have been impossible to gather the desired information. The committee was able to obtain answers to 356, or 77.5 per cent, of the questionnaires regarding the 458 deaths.

BIRTHS, DEATHS AND MORTALITY RATE

During the year of the survey there were 60,424 live births recorded in the State. Of these, 458 women died, a puerperal death rate of 7.2 per 1,000 live births. The number of puerperal deaths and the death rate per 1,000 live births between 1930 and 1935 is shown in *Table 1*. During this span of five years the

Table 1

MATERNAL MORTALITY IN GEORGIA (1930-1935).



number of deaths has decreased by 200, and the death rate per 1,000 live births by 3.7.

Attendants at Time of Confinement.—A study of *Table 2* shows that 57 per cent of the 60,424 live births were attended by physicians or interns. This left 43 per cent of the confinements to the care of midwives or other unskilled attendants. Physicians attended 86 per cent of the births among white women and 21 per cent of the negro births. Midwives, almost a necessity in Georgia rural communities, attended only 14 per cent of the white births, but shouldered the responsibility of 79 per cent of the negro births. It would be of interest to learn of the average financial compensation which they received in attending these 19,281 negro women.

Table 2

ATTENDANTS AT TIME OF CONFINEMENT DURING 1935

	Total	Per Cent	White	Per Cent	Colored	Per Cent
Live births	60,424		35,634		24,790	
Attended by physicians	35,701	57	30,322	86	5,379	21
Attended by midwives	24,482		5,201	14	19,281	79
Stillbirths	3,968		1,524		2,444	

Maternal death rate of 7.2 per 1,000 live births.
Infant death rate of 69 per 1,000 live births.

Under the present set-up for maternal care in our State the midwife is a necessity. She cares for an enormous number of confinements among patients not often desired by physicians, because of their inability to pay for his services. Would it not pay the medical profession to encourage their presence? The author believes that it would and that a new system should be worked out by which midwives could be better prepared, properly supervised and instructed from time to time. They certainly relieve the rural practitioner of a great bulk of undesired obstetrical clientele. A glance at *Table 13* will show their responsibility in maternal mortality during the year of this survey.

Causes of Death.—An effort was made to follow the international list of the causes of death in this report. Thus, the results can be compared with studies in other states.

As noted in *Table 3*, albuminuria and eclampsia were responsible for 123 or 34.5 per cent of the 356 deaths. Septicemia was next, and was the cause of 93 or 26.1 per cent. Infection leads the list as the cause of death if mortalities due to septic abortions are combined with those due to sepsis occurring after the period of viability of the fetus. Hemorrhage,

exclusive of those accompanying abortions and ectopic gestations, caused 14.9 per cent of the deaths. Accidents of labor were responsible for only 2.8 per cent of the fatalities. In our 1933 survey the accidents of labor made up 7.3 per cent of the deaths.

Table 3

PERCENTAGE DISTRIBUTION OF 356 DEATHS BY PRINCIPAL CAUSES

Cause of Death	Number	Per Cent
Abortion, sepsis	49	13.7
Abortion, hemorrhage	4	1.2
Septicemia	93	26.1
Albuminuria and eclampsia	123	34.5
Accidents of labor	10	2.8
Hemorrhage	53	14.9
Ectopic gestation	7	1.9
Vomiting of pregnancy	4	1.2
Non-obstetrical	13	3.7

Of the fifty-three deaths due to abortion, 76 per cent were residents of urban centers; 47 per cent were married women; 61 per cent were credited with having been criminally induced; 94 per cent died of sepsis; 72 per cent died in their homes and 9 per cent had a curettement as a part of their treatment. Abortion caused 10 per cent of the deaths among white women, and 17 per cent of those among Negroes.

The niney-three deaths due to septicemia were about equally divided among Negro and white women; 37 per cent were delivered by physicians; 63 per cent were attended by midwives or others; 58 per cent were multiparae; 26 per cent were delivered by some form of operative procedure; 47 per cent died in their homes, and only 6 per cent had the advantage of blood transfusions. Eighty-three per cent of the babies born of mothers dying of sepsis lived. It was the primary cause of death in 17 per cent of the eclamptics, and 30 per cent of the deaths among women whose pregnancies were complicated by placenta previa.

Of the twenty-six deaths due to placenta previa, 76 per cent were among multiparae; 60 per cent had reached the eighth month of gestation; 61 per cent lived in rural districts; 78 per cent of the women gave a history of one or more warning hemorrhages prior to the one which proved fatal; 9 per cent had the advantage of blood transfusions, and 30 per cent died of sepsis. Cesarean section was the procedure of choice in the treatment of 22 per cent. Braxton Hicks version, or version followed by immediate extraction, was the operation of choice in 52 per cent. Version was more frequent in rural than in urban centers. The fetal mortality in placenta previa was 53 per cent.

Of the 123 deaths among women whose pregnancies were complicated by eclampsia, 57 per cent were among white women and 43 per cent among Negroes; 86 per cent were less than 26 years old; 73 per cent had received no prenatal care; 86 per cent had reached the eighth month of gestation; 19 per cent died undelivered; 33 per cent were delivered by operative procedures and 55 per cent died in their homes. The fetal mortality was 45.6 per cent.

Distribution of Deaths Among Whites and Negroes.—Table 4 reveals that 188, or 52.8 per cent, of the deaths were among white women. Evidently the presence of a large Negro population is not one of the outstanding causes of an increased maternal mortality rate in our State. The incidence of death due to abortions was 6.7 per cent higher among Negroes than among white women. Hemorrhage caused more deaths in white women by a ratio of two to one. Deaths due to eclampsia and septicemia were about equally divided between the two races.

Table 4

CAUSE AND PERCENTAGE DISTRIBUTION OF DEATH IN WHITES AND NEGROES

Cause of Death	Total		Whites		Negroes	
	Number	Per Cent	Number	Per Cent	Number	Per Cent
Total	356	100.0	188	52.8	168	47.2
Abortion, sepsis	49	13.7	20	10.7	29	17.4
Abortion, hemorrhage	4	1.2	3	1.7	1	.7
Ectopic gestation	7	1.9	2	1.1	5	2.9
Septicemia	93	26.1	47	25.0	46	27.5
Hemorrhage	53	14.9	37	19.8	16	9.5
Aibuminuria and eclampsia	123	34.5	64	34.1	59	35.1
Vomiting of pregnancy	4	1.2	2	1.1	2	1.2
Accidents of labor	10	2.8	4	2.2	6	3.4
Non-obstetrical	13	3.7	9	4.3	4	2.3

Deaths According to Race and Marital Status.—As noted in *Table 5*, of the 188 white women who died, 13, or 3.5 per cent, were illegitimately pregnant; of the 168 deaths in Negroes, 40, or 11.5 per cent, were not married. Of the 53 illegitimately pregnant women, 33, or 62.2 per cent,

Table 5

DEATHS ACCORDING TO RACE AND MARITAL STATUS

Race	Married		Single	
	Number	Per Cent	Number	Per Cent
White	175	49.1	13	3.5
Negro	128	35.9	40	11.5

Table 6

RELATION OF PRENATAL CARE TO CAUSES OF DEATH
(Exclusive of 60 Abortions and Ectopics)

Cause of Death	Deaths		Adequate		Inadequate		None	
	Number	Per Cent	Number	Per Cent	Number	Per Cent	Number	Per Cent
Total	296	100.0	52	17.5	42	14.2	202	68.3
Premature Sep. Plac.	4	1.3	1	1.9	0	0.0	3	1.4
Placenta previa	26	8.7	6	11.5	4	9.5	16	7.9
Postpartum hemorrhage	23	7.7	5	9.6	3	7.1	15	7.5
Sepsis	93	31.4	17	32.8	11	26.2	65	32.2
Albuminuria and eclampsia	123	41.9	16	30.7	19	45.2	88	43.7
Accidents of labor	10	3.4	2	3.8	2	4.8	6	2.9
Vomiting of pregnancy	4	1.3	1	1.9	1	2.4	2	.9
Non-obstetrical	13	4.3	4	7.8	2	4.8	7	3.5

died as a result of abortions. Twenty-six of these were credited with having been criminally induced.

Prenatal Care.—In studying *Table 6*, after excluding 60 women whose death was due to abortion and ectopic pregnancy, there remained 296 women who should have received prenatal care. Of these 296 women, 202, or 68.3 per cent, had had no prenatal examination or instruction from a physician. In the 1933 Georgia survey this figure was 81.1 per cent.

The Committee followed the attending physician's statement for a determination of the amount of prenatal care that the women had received. Evidently there is a misunderstanding on the part of the medical profession of what constitutes adequate prenatal care. Notice that 16, or 30.7 per cent, of the 52 women credited with adequate prenatal care died of albuminuria and eclampsia. Prenatal care, such as it was, was much more frequent among white than among Negro women, and in both groups it was more frequent in urban than in rural areas.

Table 7

COMPARATIVE DANGER OF FIRST AND SUBSEQUENT PREGNANCIES BY CAUSE OF DEATH

Cause of Death	Total	Primagravidae		Multigravidae	
		Number	Per Cent	Number	Per Cent
Total	356	173	48.6	183	51.4
Abortions	53	18	10.5	35	19.1
Ectopics	7	2	1.2	5	2.8
Hemorrhage	53	11	6.4	42	22.9
Sepsis	93	38	21.9	55	30.1
Album. and eclamp.	123	91	52.5	32	17.5
Pernicious vomiting	4	3	1.7	1	.6
Accidents of labor	10	6	3.4	4	2.1
Non-obstetrical	13	4	2.4	9	4.9

Parity.—The relationship of parity to death is shown in *Table 7*. One hundred and seventy-three, or 48.6 per cent, were primigravidae, and 183, or 51.4 per cent, were multigravidae. Albuminuria and eclampsia were responsible for deaths more often in primigravidae than multigravidae by a ratio of three to one.

Abortions caused 10.5 per cent of the deaths in nulliparae and 19.1 per cent of the fatalities among multiparous women. Hemorrhage as a cause of death was approximately four times more frequent among multigravidae than in primigravidae, the percentage column showing 22.9 per cent versus 6.4 per cent. Septicemia deaths were slightly higher among multiparae.

Period of Gestation.—The relation of the duration of pregnancy to the various causes of death is shown in *Table 8*. Seventy-four, or 21 per cent, of the deaths occurred during the first two trimesters of pregnancy, and 222, or 79 per cent, died in the third trimester. Two deaths were due to peritonitis following laparotomies in the fifth and seventh months, respectively, for the removal of secondary abdominal pregnancies.

Operations.—Of the 282 women who died in the third trimester of pregnancy, 108, or 34.1 per cent, had some form of operative procedure directed toward delivery before death. These include unsuccessfully attempted operative procedures as well as completed operations and are

Table 8

CAUSE OF DEATH BY MONTH OF PREGNANCY

Cause of Death	Total	Months						
		1-3	4	5	6	7	8	9
Abortion, septic	49	32	11	4	2			
Abortion, hemorrhage	4	2			2			
Ectopic pregnancy	7	5		1		1		
Placenta previa	26				1	2	15	8
Premat. separation	4					1	1	2
Postpartum hem.	23					2	1	20
Puerperal sepsis	93					3	12	78
Album. and eclamp.	123			2	2	10	26	83
Pernicious vomiting	4		3	1				
Accidents of labor	10							10
Non-obstetrical	13	1	2	1	3	3		3
Total	356	40	16	8	10	23	55	204

recorded in *Table 9*. There were 25 deaths following cesarean sections. Forty-four deaths occurred after forceps deliveries; 29 died following versions; 5 died after breech extractions. Note that 40, or 33 per cent, of a total of 123 cases of albuminuria and eclampsia were delivered by operative procedures.

Table 9

RELATION OF METHOD OF DELIVERY TO CAUSE OF DEATH IN 108 OPERATIVE DELIVERIES: TYPE OF DELIVERY

Cause of Death	Total Operative Deliveries	Multiple Methods	Cesarean	Forceps	Version	Breech	Hysterectomy
Total	108	3	25	44	29	5	2
Premat. sep. of plac.	3		1	2			
Placenta previa	22	1	4	3	13	1	
Postpartum hem.	11			9	2		
Sepsis	24	1	6	7	7	1	2
Album. and eclamp.	40	1	14	18	6	1	
Accidents of labor	8			5	1	2	

Methods of Delivery of Placenta.—It was found that the placenta was manually removed from the uterus of forty-two, or 13.7 per cent, of 306 women over twenty-eight weeks pregnant. Postpartum hemorrhage was responsible for 15 and sepsis for 27 of the deaths following manual removal of the placenta.

Anesthesia.—A study of *Table 10* shows that in 246 women reaching the third trimester of pregnancy, 101, or 41 per cent, were delivered without the use of anesthesia. Four deaths as a result of spinal anesthesia were studied. All of these died on delivery or operating tables.

Table 10

TYPE OF ANESTHESIA USED IN THE DELIVERY OF 246 CASES IN THE THIRD TRIMESTER

Anesthesia	Number	Per Cent
Chloroform	46	18.7
Ether	70	28.5
N_2O or ethlene	24	9.8
Local	1	.4
Spinal	4	1.6
None	101	41.0

Methods of Onset of Labor.—Table 11 reveals that the onset of labor was spontaneous in 229, or 89.9 per cent, of the women who were in labor in the third trimester of pregnancy. In spite of the fact that artificial rupture of the membranes is considered safer, more reliable and easier, it was found that other methods were resorted to three times more often than this simple method of inducing labor.

Table 11

METHOD OF ONSET OF LABOR IN WOMEN REACHING THE THIRD TRIMESTER

(Exclusive of Cesareans and Deaths Prior to the Onset of Labor)
255 Cases

Methods	Number	Per Cent
Spontaneous	229	89.9
Induction:		
Castor oil and quinine	2	.7
Pituitrin	2	.7
Art. rupture of membranes	5	1.9
Catheter	9	3.6
Bag	3	1.2
Packing	2	.8
Combination	3	1.2

Cesarean Section.—Included in the survey are twenty-five deaths following delivery by cesarean section. The indications are listed in *Table 12*. Several other deaths followed cesarean sections, but the Committee was unable to secure

Table 12

PRINCIPAL INDICATION FOR CESAREAN SECTION AND IMMEDIATE CAUSE OF DEATH: 25 CASES

Indication	Total	Cause of Death		
		Sepsis	Toxemia	Hemorrhage
Eclampsia	14	12	2	
Placenta previa	4	2		2
Obstructed labor	5	5		
Transv. presentation	1	1		
Premat. sep. of plac.	1			1

answers to its questionnaires regarding these deaths. Of the twenty-five cases studied, twenty were whites and five were Negroes. Seventeen were primigravidae. All were emergency operations, with the exception of four cases. Twenty-three were classical sections and two were Porro operations. All of the operations were performed in hospitals. In six, an attempt had been made at delivery from below prior to the performance of delivery by the abdominal route.

Eclampsia was the indication for fourteen of the sections; placenta previa for four; obstructed labor for six, and abruptio placentae for one. Peritonitis was the cause of 80 per cent of the deaths following cesarean sections.

Attendant at Delivery.—Table 13 indicates the attendants at 282 labors in women after the twenty-eighth week of gestation. Fourteen, or 4.9 per cent, of the deliveries followed by death were attended by others than midwives or physicians; midwives alone attended fifty-five, or 19.3 per cent; midwives were replaced or relieved by physicians in seventy-one, or 25.3 per cent; and 142, or 50.5 per cent, were attended by physicians alone.

Table 13

ATTENDANT AT DELIVERY BY CAUSE OF DEATH
282 CASES

(Pregnancies of 28 weeks and over)

Cause	Total	Physician or Intern	Midwife	Midwife Relieved by Physician	Others
Total	282	142	55	71	14
Per cent	100	50.5	19.3	25.3	4.9
Premature separation	4	3		1	
Placenta previa	26	14	2	8	2
Postpartum hemorrhage	23	10	5	7	1
Sepsis	93	35	34	18	6
Albumi. and eclampsia	116	67	9	37	3
Accidents of labor	10	6	4		
Non-obstetrical	3	2			1

Remember that in *Table 2* midwives were shown to have attended 43 per cent of the total live births reported in the State during the year 1935.

Place of Delivery and Place of Death.—Of the 356 deaths studied, 33 per cent were among women of urban centers. Therefore, 67 per cent or more than two-thirds of the deaths were among women who had lived in rural districts. Unfortunately, 54.2 per cent of the women died in their homes without the advantage of hospitalization. Of those who died in hospitals, only 6.7 per cent had planned hospitalization at the time of confinement. The greater per cent of the emergency admissions had been delivered of the fetus when admitted to hospitals.

COMMENTS

The fact that 34.5 per cent of the deaths were among women suffering with eclampsia should cause us to realize that a serious situation exists in regard to the type of maternity care that the women of our State receive during pregnancy. There is a need for the development of prenatal centers throughout the State. These centers should be under the supervision of conscientious and competent attendants.

Good prenatal care would have prevented the loss of a large number of the eclamptic women, as well as a large per cent of those dying of the hemorrhages associated with pregnancy. There is especially a need for the adoption of a more uniform, conservative treatment for eclampsia. It would pay big dividends in the form of maternal lives.

The incidence of death due to abortion is too high. The public should be taught and impressed with the danger associated with abortions.

A more careful observation of the principles of asepsis and antisepsis, along with fewer vaginal examinations and manipulations through an unprepared vulva during labor, should lower the incidence of deaths due to sepsis.

An educational program, informing the public of the necessity of prenatal care and medical supervision throughout pregnancy and labor, should soon cause the laity to cooperate with the medical profession in its effort to reduce maternal mortality. The public is not yet conscious of the potential dangers associated with the process of reproduction. It is the duty of the medical profession to inform it.

Also on the part of the medical profession, there is a need for better postgraduate training. New knowledge and advancement in the science and art of obstetrics should be placed in the hands of the multitude of medical attendants responsible for the conduct of our mothers throughout pregnancy and labor.

The midwife, attending almost one-half of the births in our State, cannot be neglected.

She should be better instructed, and her work more carefully supervised.

The problem of reducing maternal and infant deaths is a universal one, and only by the conscientious and persistent efforts of the public, the medical profession and the health authorities can it be reduced.

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A STUDY OF THE VAGINAL FLORA IN THE NORMAL FEMALE

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Numerous studies[1 to 36] of the bacterial content of the vaginas in obstetric and gynecologic patients have been made. The results reported in this paper represent an effort to establish for our own patients in the obstetric and gynecologic dispensaries a basis for applying to them the terms "normal or abnormal vaginal flora." In an effort to determine the bacterial contents of the vagina of both obstetric and gynecologic patients with obvious disease of the vulva, vagina and cervix, in patients with such diseases as pellagra, sprue, diabetes, fungus infection and obvious endocrinopathies and in patients seen for sterility study we found that our conception of the normal vaginal flora was most faulty and irregular. To determine the actual organisms of vaginas at any one time does not offer insurmountable problems of bacteriologic technic when procedures for obtaining the material and methods of smearing and culturing this material are standardized. To analyze the bacteriologic findings many conditions have to be considered before the interpretations of the findings may have a consistency worthy of clinical consideration or application. The methods of obtaining the smears and cultures, the sanitary habits of the patient, the season of the year in which the material is obtained, the time relationship between the taking of the cultures and menstrual flow and coitus, the duration of pregnancy, the use of baths, douches, vaginal antiseptics, the general metabolic and endocrinologic condition of the patient are but a few of the conditions which have to be considered before dogmatic rules of diagnosis and therapy may be used. It is also of extreme importance that the medical student be given the opportunity to look upon the obstetric and gynecologic patient, suffering from vaginal disease, as one upon whom bacteriologic diagnostic methods, other than the routine smear and hanging drop, should be employed.

SOURCE OF MATERIAL FOR SMEARS AND CULTURES

Bacteriologic studies were made of the vaginal secretions of 114 "normal" pregnant women and of the vaginal secretions of 100 "normal" gynecologic patients. The term "normal," as applied to these dispensary patients, meant: (1) that all patients in either the obstetric or gynecologic group had no metabolic or endocrinologic abnormalities evident on physical examination; (2) that all patients were free of evidence of abnormal discharges from the vagina and had no signs of vulval, vaginal or cervical disease; (3) that all patients had no evidence of recent abortion and no signs of uterine or cervical bleeding; (4) that all patients showed smears negative for gonococci and hanging drop preparations negative for trichomonads; (5) that none had been receiving any form of vaginal therapy, as douches, tampons or suppositories.

In Döderlein's original work published in 1892 he divided the vaginal secretions of the pregnant woman into two types, the normal and the pathologic, on the grounds of naked eye appearance, bacteriology and cytology, and on the reaction of the secretions to litmus paper. As noted by Logan,[28] this arbitrary division has been questioned by other observers. It has been shown that the vaginal secretion in normal women is not always strongly acid to litmus paper, that it may contain pus cells and that its flora is by no means always restricted to Döderlein's bacillus and yeasts. Logan found on the study of smears of clinically normal vaginas that epithelial cells and polymorphonuclear leukocytes were almost always present in small numbers. We confirmed these findings and unless the pus cells were markedly increased in number the patient was included in the series of "normal" clinical vaginas.

METHOD OF OBTAINING SMEARS AND CULTURES

In the early part of the work efforts were made to give the external genitalia elaborate aseptic preparation and care. It was soon evi-

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dent, however, that the use of solutions gave greater chances for carrying the organisms of the external genitals into the vagina. In the pregnant group it was quite simple to spread the labia and the introitus and to insert the swabs into the vagina without contamination. In the gynecologic group it was often necessary to use sterile specula to obtain a view of the upper vagina for smear and culture material. The specula were never introduced to the full length of the vagina and smears were taken from the vaginal walls on each side and below the cervix. Any contact of swab with the speculum, lower vagina or cervix was considered contamination and the patient was excluded from the study.

REACTION OF UPPER VAGINAE

Following the work of Logan[28] the reaction of the upper vagina was tested roughly in half of the patients, but was discontinued in that we could find no consistent relationship between the reaction of the vagina and the presence or absence of Döderlein's bacillus and the presence or absence of streptococci.

CULTURAL METHODS

Before this work was begun, methods of culture were sought that would give a fair degree of correlation between smear and cultural findings. While we do not feel that this has been accomplished, we do feel that with some experience one can predict from smear study the predominating organisms which will be encountered.

Solid media were given preference over primary broth cultures in order to obtain an approximation of the number of viable organisms present in the original material. In this way we learned that while Staphylococcus albus was present in a large percentage of the cases, rarly if ever could it be termed the predominating organism. In many cases only six or more colonies were present on the plates. This percentage could be lowered considerably if better methods were available for the collection of the cultures.

The culture media used and the methods of culture are shown in the accompanying illustration (*Fig. 1*). If any degree of correlation is to be obtained, anaerobic as well as increased CO_2 tension methods are essential. Dextrose or some form of carbohydrate is necessary for the growth of a large majority of strains of Döderlein's bacillus, and the addition of dextrose favors the growth of the anaerobic types of cocci. No dextrose in the aerobic plate obviated the necessity of transplantation for study of hemolysis, pigment formation and so on.

Theoretically the eosin methylene blue plate and the Sabouraud's agar slant might be dispensed with, but gave in many instances the quick isolation of fungi and organisms in the gram negative bacilli groups.

Many strains of diphtheroids, as well as streptococci of the gamma type, were micro-aerophilic on isolation. We feel sure that the percentages of these two groups would have been considerably lower had not this type of culture been used.

In the beginning efforts were made to classify

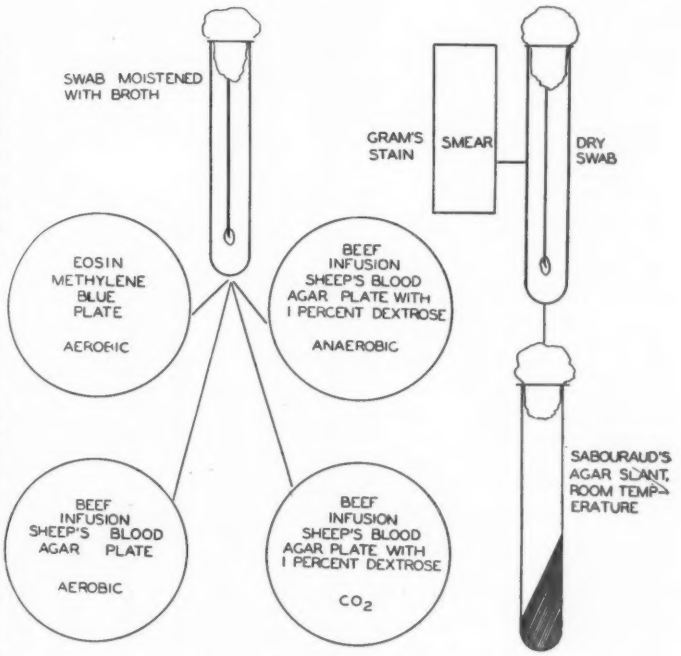

Fig. 1
Culture media used and methods of culture.

the anaerobic group of cocci using cultural characteristics such as gas formation and odor. This was soon abandoned due to our inability to subculture many strains, particularly the Type B of Colebrook. All strains were either inoculated into deep shake glucose agar stabs to demonstrate anaerobiosis or failed to grow when transplanted to aerobic slants. Most of the colony types fitted readily into the classification proposed by Colebrook, Types A and B usually occurring together. Rarely Type B occurred alone. No hemolytic types were encountered and only on two occasions was the black-pigment producing type of Colebrook seen. In the Type A group several apparently different types were observed, namely: white colonies, with a tendency to heap up, and a second type that appeared flatter, of darker color and with definite raised central papillae. Much work is to be done before a final and satisfactory grouping of these organisms will be possible.

RESULTS

The results of the work are given in *Fig. 2*. The percentage incidence of the various organisms is graphically presented.

Of diphtheroids, the predominating group of organisms encountered in both types of patient, little can be said. That they are present normally in the vagina has been found by all investigators. Any attempt at classification is difficult, as the number of species far outstrips our present knowledge of bacteriology. Anaerobic and micro-aerophilic strains were encountered as well as aerobic and all are included in one group.

In the *gamma* streptococcus group are included strains that were micro-aerophilic on the primary isolation plates but proved aerobic on isolation. All of these strains were tested after isolation for hemolysis on standard blood agar plates and are placed in one group. All strains producing a green discoloration of the agar and secondary zone of hemolysis were placed in the *alpha* group. Many strains of diphtheroid bacilli were encountered that produced *alpha* hemolysis on blood agar and consequently some strains of *alpha* streptococci may have been overlooked.

All strains of gram negative bacilli were identified by using carbohydrate fermentations and the usual routine laboratory procedure.

Three strains of streptococci with the *beta* type of hemolysis on blood agar plates were encountered. All three of these strains were in the

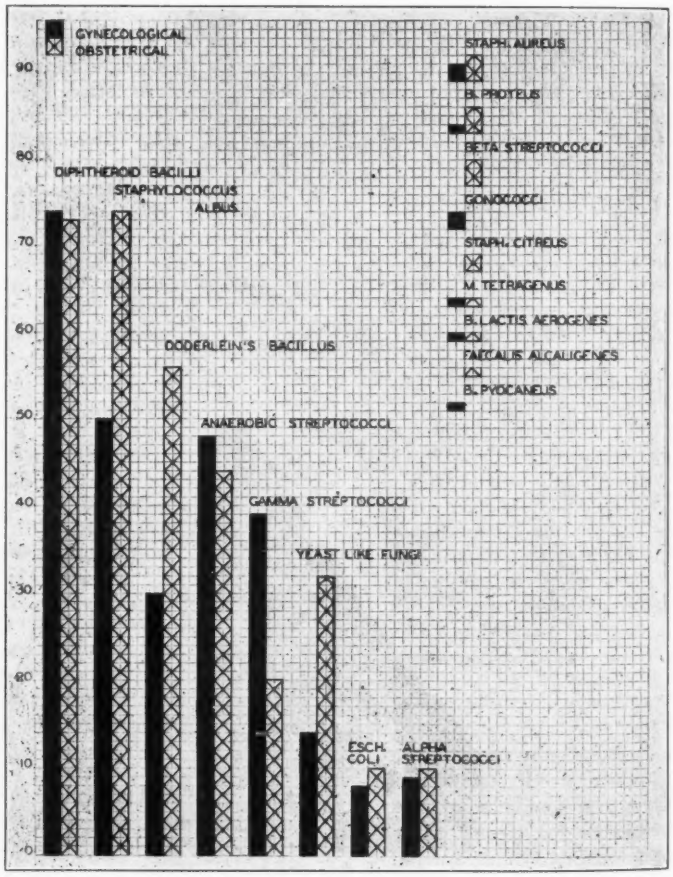

Fig. 2
Percentage incidence of various organisms.

pregnant series. Two strains failed to produce soluble hemolysin and were classed as pseudo-hemolytic. Recent work by Lancefield,[33] who used a group precipitin reaction to differentiate between the Type A, or human infectious group, and groups of animal origin, and by others using Lancefield's technic, indicate that the organisms of Type A are rarely encountered in the vagina in the absence of infection. Hare,[34] in England, separated Type A from other types in the vagina by the bactericidal action of the patient's blood as well as blood of normal patients. Our one strain was not considered of sufficient importance to test serologically using Lancefield's method. It is interesting to note that Logan[28] found no frankly hemolytic streptococci in his series and that Lockhart[14] found only 1 per cent in 100 patients. Burt White and Armstrong,[18] using anaerobic as well as aerobic methods, isolated hemolytic streptococci from 22 (14 per cent) of 153 patients. As suggested by Logan, this high figure, as contrasted with other reports, probably means that some of the organisms of the non-hemolytic or pseudo-hemolytic group were included.

In the clinical application of the bacterial findings from the vaginas of pregnant women it has been customary to divide patients into groups as was done by Schroder[11] in 1921. He divided his patients into three types, a grouping claimed by him to correspond to the grade of cleanliness of the patients.

Group 1—Only Doderlein's bacillus present in vagina.
Group 2—Doderlein's bacillus present in small numbers, other types of organisms predominating.
Group 3—No Doderlein's bacillus.

In our own results we have included in Group 1, in addition to Döderlein's bacillus, Staphylococcus albus and yeast, because in many instances the vaginal bacilli and yeast or vaginal bacilli with a few colonies of staphylococci were alone present. Our Groups 2 and 3 correspond to the groups of Schroder.

DIVISION OF FLORA INTO TYPES

	Obstetric	Per Ct.	Gynecologic	Per Ct.
Type 1—Vaginal bacilli alone or vaginal bacilli with either Staphylococcus aibus or yeast	19	16.66	15	15
Type 2—Vaginal bacilli with organisms other than the two named above	45	39.47	15	15
Type 3—No vaginal bacilli	50	43.85	70	70.0
	114		100	

In contrasting the findings in the colored and white obstetric and gynecologic groups we find the following percentages of incidence of the various organisms:

GYNECOLOGIC PATIENTS

Organism	Colored Per Cent	White Per Cent
Diphtheroid bacilli	74	74
Staphylococcus albus	52	48
Anaerobic streptococci	54	46
Gamma streptococci	32	44
Alpha streptococci	8	10
Esch. coli	8	10
Yeast	14	14
Doderlein's bacillus	22	36

OBSTETRICS

Diphtheroid bacilli	74	70
Staphylococcus albus	78	61
Anaerobic streptococci	49	30
Gamma streptococci	20	21
Alpha streptococci	10	12
Esch. coli	10	12
Yeast	36	21
Doderlein's bacillus	49	73

Schottmuller,[5] Schwarz and Dieckmann[17 19] have shown the importance of the anaerobic streptococci in causing puerperal fever. This group also constitutes a definite hazard in pelvic operations on gynecologic patients. In our obstetric groups the organisms were found in 44 per cent of the patients. The colored patients showed an incidence of 49 per cent, the white patients an incidence of 30 per cent. In the gynecologic series anaerobic streptococci were found in the secretions of 43 per cent of all patients. Fifty-four per cent of the colored patients and 46 per cent of the white patients had the organisms in the vaginal secretions.

It would seem logical to institute therapy prior to operation on this gynecologic group and to test such therapy by an intensive bacteriologic study.

In a recent publication Weinstein, Bogin, Howard and Finklestone[38] have stressed the importance ascribed to Döderlein's bacillus in the production of the acidity of the vaginal tract and also the role played by vaginal acidity in the prevention of infection of the vaginal tract, especially during pregnancy. The question asked is whether this increased growth of Döderlein's bacillus in the vaginal tract of pregnant women is due to increased hydrogen ion concentration or to some other factors. They found an increasing incidence of Döderlein's bacillus in the vagina as they progressed from examinations of prepubertal children to examinations of menstruating women and of non-gravid and gravid women. They suggest that since estrin is found in only small quantities in prepubertal children, in increasing amounts in women as menstruation comes on and in its highest concentration in the

pregnant women, and especially in the later months of pregnancy, that estrin increases the vaginal acidity and also increases the growth of Döderlein's bacillus. They quote the work, as yet unpublished, of Hall and Lewis and of Lewis and Weinstein, which shows that the administration of estrin to monkeys increases the vaginal acidity and that Döderlein's bacilli often reappeared in the vagina after estrin administration. In short, the natural or artificial introduction of estrin favored the presence of the vaginal bacillus and similarly a low pH or increased acidity inhibited the presence of gram-negative rods, diphtheroids and gram positive cocci.

The work of Lewis[39] and TeLinde and Brawner[40] in the treatment of gonococcal vaginitis in children by the use of theelin and amniotin had given to us the problem of applying to a study of the vaginal flora a study of the flora before and after such administrations of endocrinal preparations. We were also interested in attempting to find a method to determine the glycogen content of the vaginal mucosa in the "normal" and the diseased vagina. At the present time in a few patients we have not been able to influence the vaginal flora in "normal" patients by the use of these endocrine preparations or to prove a satisfactory method of ascertaining from the study of biopsy specimens of the vagina of women just what the glycogen content of the vaginal mucosa approximates because of the difficulty in interpretation of the glycogen stains.

SUMMARY

(1) An effort has been made to find an accurate method of study of the vaginal secretions of "normal" obstetric and gynecologic patients using a cultural method which gives a fair correlation between smear and cultural findings and which gives importance to the incidence of fungi and anaerobic organisms.

(2) The results support the work of numerous observers that Döderlein's bacillus is found in much higher percentage in the obstetric group as contrasted to the gynecologic group. We had expected that as Döderlein's bacillus and yeast increased in each group, the incidence of anaerobic streptococci and of *gamma* streptococci would fall. In our series the anaerobic streptococci were little influenced by the presence of Döderlein's bacillus or yeasts, but the *gamma* streptococci showed a very definite fall in those patients with the higher percentages of Döderlein's bacillus and yeasts.

(3) Staphyloccocci were consistently found, but were not the predominating organisms.

(4) Colon bacilli in our series were found in higher percentages than in the work of other observers.

(5) *Alpha* streptococci were found in low percentages.

(6) *Beta* streptococci were found in only 2 of 114 obstetric patients, an incidence which adheres closely to the reports in the literature.

(7) Work is now in progress on the incidence and classification of the vaginal fungi; on the vaginal flora in pellagra, sprue, diabetes; on patients before and during pregnancy and the puerperium; and on patients who are being examined and treated for sterility or endocrinologic disorders.

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DISCUSSION (*Abstract*)

Dr. Richard W. TeLinde, Baltimore, Md.—The foundation upon which the study of all pathological conditions should rest is a thorough knowledge of normal anatomy, histology, physiology and bacteriology. The greatest reason why our efforts in treating such common gynecological conditions as functional bleeding and dysmenorrhea have so largely failed is our very incomplete knowledge of the physiology of menstruation. Drs. Carter and Jones have contributed something to the foundation work which we must have before we can progress with our knowledge of pathological conditions of the vagina. Fortunately, in the past few years there has been a real interest in working out the etiology and treatment of vaginitis, a condition which has been for the most part poorly understood and miserably treated.

Take for example the so-called trichomonas vaginitis. We presume that the vaginalis Trichomonas is responsible for this condition because we see this flagellate frequently associated with a rather typical clinical picture. But at times we see a clinical picture which I cannot distinguish from the vaginitis associated with the flagellate in which no flagellates can be found. Then, too, we find the flagellate in a fair percentage of women (as high as 20 per cent in pregnant women) which have no evidence of vaginitis and finally, all attempts at producing the disease by the introduction of trichomonas into the vagina have failed. The frequent association of the streptococcus with the trichomonas has caused some to consider it the real etiological agent. The matter is far from settled and one reason is that our incomplete and inexact knowledge of the normal vaginal flora does not permit us to evaluate the findings in this pathological condition.

The question has been raised as to whether the presence of estrin in the tissues increases the incidence of the Döderlein bacillus. In Dr. Brawner's and my studies on gonococcal vaginitis treated with estrin, we were not impressed with this. It is true that in some cases the Döderlein bacillus did appear after the gonococcus had disappeared and the vagina was well under the influence of estrin. But this organism did not appear with any regularity and in many instances streptococci and other organisms seemed to replace the gonococcus. I do not believe it is necessary to consider the acidity produced by Döderlein's bacillus to be the factor which finally eliminates the gonococcus. Certainly many of our cases of gonorrheal vaginitis got well without the appearance of the Döderlein bacillus in the smears.

There is one point in particular in Drs. Carter's and Jones' paper which deserves emphasis from the standpoint of the practical obstetrician and gynecologist. I refer to their finding of the anaerobic streptococcus in nearly 50 per cent of the vaginas cultured. Schwarz and Dieckmann, Harris and Brown, and T. K. Brown, who have done careful bacteriological studies of the puerperally infected uterus, all agree that the anaerobic streptococcus is responsible for puerperal infection in a large percentage of cases. These organisms are believed by these observers to be of endogenous origin and the finding of them in the vagina in a high percentage of cases by Drs. Carter and Jones makes this assumption highly probable. It also stresses the importance of a thorough vaginal clean-up before all gynecological operations in which the vagina is to be entered. This applies to even the lowly dilatation and curettage. The cervical canal, particularly in parous women, is open to the vaginal flora. No matter how thoroughly the vaginal toilet, the cervical canal may still harbor organisms and it has been my routine to swab out the canal with a cotton pledget soaked in alcohol before putting either a curette or a Rubin's cannula into the uterine cavity.

Dr. Karl John Karnaky, Houston, Tex.—I have been studying the vaginal flora for the past five years and can say that the more I study it the less I know about it.

It is of interest to study the vaginal flora and the condition of the epithelial cells in a fresh smear under the high and low power of the microscope on all cases coming to the gynecological and obstetrical clinic and correlate these findings with the pathology found in the vagina.

One should also correlate the pH of the secretion with the bacterial flora and should compare sections of the mucous membrane to see the height of epithelial cells, the glycogen and microscopic appearance in a vagina with and without a discharge.

We know that a vaginal discharge is a mixture of fluid, pus cells, and epithelial cells in all stages of degeneration, that is débris and bacteria. Sometimes we see an excess of secretion with no epithelial cell degeneration and no bacteria other than the Döderlein. This

is an excess of the normal secretion, which we should be able to recognize. My bacteriological findings are about the same as those reported by Drs. Carter and Jones.

In my 4,000 cases of trichomas vaginitis, the staphylococcus was the most common associated organism. *Streptococcus* hemolyticus was not found so often as is reported by other investigators. It was rarely found.

There is a close relationship between the bacterial flora, the pH or acidity, the glycogen content of the mucous membrane, and the height of the vaginal epithelial layer. This is especially true in vaginal leukorrheas.

The glycogen of the mucous membrane passes into glucose in the vagina and finally changes into lactic acid. This acid produces a pH in the vagina of approximately 4.0, the normal pH. When the vagina is at this pH, there is rarely any pathologic condition. At this pH, the most common organism found is the Döderlein bacillus. *Monilia albicans* will also live at this pH, but prefers other hydrogen ion concentration around pH 5.5.

Just as soon as the acid pH of the vagina begins to approach the alkaline side, the vaginal flora becomes more mixed, that is, vaginal infections create a hypo-acidity of the vagina or hypo-acidity favors their growth. Most pathogenic organisms grow at a pH of 6.0-7.6.

Then on the other hand, as one makes the vagina more acid (lower pH) the vaginal flora becomes less mixed until we get a sterile vagina or one with no Döderlein bacilli or acid requiring micro-organisms. So what I want to say is that alkaline douches are not indicated as therapy. The reason why soda douches help is that they dissolve the cervical secretion, which is also alkaline, and so long as the patient uses the douche, there is no discharge because it has been washed out, but the patient is not cured. But we all know the treatments for cervical infection are conization, cautery, or coagulation. An acid douche (pH 4.0) will in some cases help heal a cervical erosion while soda douches tend to make a vaginal leukorrhea and cervical leukorrhea. Acid (pH 4.0) causes an epithelialization (hormone reaction) and will in many cases cure cervical erosions by an overgrowth of epithelial cells.

All vaginal infections are on the acid side, with rare exceptions, and cervical infections are always on the alkaline side.

Dr. TeLinde mentions vulvo-vaginitis and treatment with estrin suppositories. I should like to describe my method of treatment, namely by acidifying the vagina in children. In April, 1935, I noted that the cure of gonorrheal vulvo-vaginitis was due more to the change in the vaginal flora than to the height of the epitheliel cells.

I previously noticed that I did not cure gonorrheal vulvo-vaginitis in children until I obtained a vaginal pH of 5.5 or below. I knew that I could keep the vagina at any desired pH by the use of chemicals and an electro-pH-determinator better and more accurately than by the use of hormone.

I tried tablets at pH of 6.0, 5.5, 5.0 on down to pH 3.3-3.0, and I found that at pH 3.0-3.3 I not only cured gonorrhea but obtained a tissue reaction in the vaginal mucosa similar to that obtained by the use of theelin. My results were so similar with the acid tablet treatment to those when I used theelin that I believe acid is what cures these cases and that acid is all we need.

A normal vagina contains a pure Döderlein flora or is sterile, high epithelial cell layer, abundance of glycogen in these cells, and a pH of 3.9-4.3.

In most pathological vaginas, we have four "hypos": hypo-acidity, hypo-Döderlein, hypo-glycogen, and hypo-epithelium.

I suggest the use of pH 4.0 douches with a few drops of menthol, minims 30 to ounces 8 (5 per cent acetic acid, ¾ tablespoonsful to two quarts of warm water) and that we use acidulated glucose-lactose tablets at pH 3.0-3.3 to restore the normal vaginal mucous membrane to its normal amount of glycogen and a pH to approximately 4.0, or normal.

THE PREVENTION OF DEATHS FROM MEASLES*

By HUNTINGTON WILLIAMS, M.D., DR.P.H.†
Baltimore, Maryland

Dr. Maurice Pincoffs, in arranging for the appearance of this paper on today's program, has done something to make clear the identic relation between the Chair of Hygiene and Public Health in the Medical School of the University of Maryland and the office of the Commissioner of Health of the City of Baltimore. This relationship was established in 1918 in the person of my predecessor, Dr. C. Hampson Jones, and continues to the present time.

It is of some interest to note that the teaching of hygiene was first incorporated in the formal curriculum of this school 103 years ago, in 1833. Robley Dunglison, an English-born physician, had been called from London in 1824 to found the Medical School of the University of Virginia. He was a prolific author and is best remembered for his Dictionary. In 1833 he joined the Medical Faculty at the University of Maryland, where he taught hygiene and other subjects. Three years later he removed to Philadelphia to become the Professor of Institutes at the Jefferson Medical College.

Hirsch,[1] Chapin,[2] Copeman,[3] Brownlee,[4] and Edward S. Godfrey,[5] Commissioner of Health of New York State, among others, have written on the epidemiology of measles. Godfrey, particularly, has pointed out the fairly regular period-

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icity of the appearance of epidemic measles, especially in urban communities, the futility of attempting to stamp out measles epidemics, the need of learning that they are true emergencies which require special handling, the extreme risk of death when measles invades an orphan asylum or a foundling hospital, and the fact that in a few months of a measles epidemic more lives may be lost among children under five years of age than can be saved by baby clinics in a year. He has shown that people do not die of measles, but of ignorance and neglect, and that the aim of the medical profession and health authorities alike should be to prevent *deaths* from measles rather than *cases*—this by guarding especially from exposure children under three years of age, in which age group the risk of measles proving fatal is greatest.

My story presents nothing essentially new, except a plea for a particular focus of attention. It can be briefly told, and best perhaps by the two accompanying tables. During the ten-year period, 1926-1935, there were 60,521 cases of measles reported in Baltimore and of these 310 died. Seventy-five and five-tenths per cent of these deaths occurred in children under 3 years of age, as appears in *Table 1.*

Table 1

MEASLES IN BALTIMORE, 1926-1935
PERCENTAGES OF DEATHS, AND CASE FATALITY RATES, BY AGES

Age Group	Percentage of Total Deaths Occurring in Specified Age Group			Case Fatality Rate or the Percentage of Reported Cases Occurring in Specified Age Group which Die		
Under 1 year	25.8	61.3	75.5	4.7	3.4	2.3
1 year	35.5			2.9		
2 years	14.2			0.9		
3 years	10.0			0.6		
4 years	2.9			0.1		
5-9	9.7			0.1		
10-14	0.6			0.1		
15-19	0.3			0.2		
20 years and over	1.0			0.3		
All ages	100.0			0.5		

The figures are based on 60,521 reported cases, of which 310 died.

The case fatality rate, or the percentage risk of dying of measles at different ages, is also shown in a striking manner. These rates are approximate, as they are based on the reported cases. For children under 1 year old with measles, 4.7 per cent die, for children under 2 years old it is 3.4 per cent, and for children under 3 years it is 2.3 per cent. However, during the years of elementary school age, the 5-14 year age group, the percentage of cases proving fatal is only 0.1.

To show it another way, and the same general characteristic is even more true of whooping cough, *Table 2* has been prepared.

Table 2

RELATIVE RISKS OF DEATH FROM MEASLES AND WHOOPING COUGH
INFANCY VERSUS SCHOOL AGE (5-14 YEARS)

Age Groups	Measles	Whooping Cough
Under 1 year	47 to 1	108 to 1
Under 2 years	34 to 1	85 to 1
Under 3 years	23 to 1	59 to 1

The figures for whooping cough are based on 40,868 reported cases, of which 837 died, in Baltimore, for the years 1921-1935, inclusive.

The table shows the relative risk of death from measles or whooping cough for children under 3 years old, by years, as compared with the risk during the school age, 5-14 years. You will see that the risk of death is 47 times greater for a child under a year old who contracts measles than for a school child, 34 times greater for a child during the first two years of life as compared with school age, and 23 times greater for a child in the first three years of life. Thereafter the risk becomes rapidly less. In whooping cough the contrast is even more marked; the risk of death is 108 times greater for a child under a year old with whooping cough than for a child of school age.

The moral of the story is quite obvious, and the duty clear: every effort should be made to keep measles away from children under 3 years old, or away from children who are poorly nourished or in poor physical condition. This is usually not difficult except in times of epidemic when it may be next to impossible. At such times young children, especially infants, should be kept at home and should be restricted as far as possible from playing with older children who have not had measles, especially if the latter are school children and therefore likely at any time to be developing the disease.

If measles attacks an infant, good medical and home nursing care may prevent a fatal outcome. Convalescent measles serum or placental extract as developed by McKhann,[6,7] if given within six days after first exposure may be of great assistance, but as has been shown,[5b]

"the main reliance in minimizing the mortality from measles must be placed on avoiding massive doses of

infection and giving adequate medical and nursing care to the sick."

Our grandmothers used to urge that all the children in a family get measles at the same time and "get it over." Then the health authorities by placards and other measures equally ineffective in controlling measles sought to prevent all spread of this most communicable of all diseases. Both were right, both were wrong. Mass prevention of measles is at present impossible. This may prove to be fortunate. If we could arrange it so that fifty years would elapse in this city without the occurrence of a case of measles and then one single case should come from a neighboring city, nearly the entire population would go to bed, as during the influenza pandemic; they would be sicker than they should and there would be great interruption of the normal life of the community.

While we have no practical agent for immunization and as long as nearly everyone is destined to have measles, the school age, 5-14 years, is the safest period in which to have it, for it is the rare exception that a case dies in that age, whereas measles is a serious danger to a child under 3 years old. The prevention of deaths from measles, therefore, is an age-specific problem of very considerable importance. It demands a thorough understanding on the part of the medical profession and the public, and an energetic effort on the part of physicians and health authorities in instructing the public in the matter, particularly during the period of a community epidemic. Such an event may be expected to occur quite regularly every two or three years in urban areas.

Finally, it would seem that any complete elimination of deaths from measles must await the development of an effective specific immunizing agent such as Jenner gave us for doing battle with smallpox back in the days when that disease was as prevalent as measles is today.

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ADRENAL HEMORRHAGE IN CHILDREN*

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This study of acute hemorrhage in the adrenal glands of children was stimulated by a puzzling surgical case in the Harriet Lane Home for Invalid Children of the Johns Hopkins Hospital. The real nature and progress of the illness of this patient were not apparent until the postmortem examination was completed. An abstract of the case record follows:

R. B. The patient was a 13-months-old infant, the second child of healthy parents. Except for constipation, he was normally healthy and was breast fed up to the age of 4½ months, at which time he began having severe diarrhea and vomiting. Breast feeding was discontinued and various formulae were tried, but the illness continued, with exacerbations, for about three months, leaving him pale and weak. He was convalescing favorably when, at the age of 8 months, he vomited blood, heaving and gagging violently. Almost a cupful was brought up. The vomitus was brown at first, but was followed by fresher looking blood. On the second day after this, one tarry stool was passed.

At the age of 11 months there was another period of vomiting for a few days, and he was taken to a hospital in Richmond for study. Routine investigation failed to reveal the cause of his gastro-intestinal upsets and bleeding.

One week before admission to the Harriet Lane Home, when the child was 13 months old, he seemed ill; his temperature was 104° for two days, and there was no apparent explanation for the fever. Three days later he suddenly became pale, exhausted and restless. The next morning he passed a stool which contained blood that was neither fresh nor tarry. The child was brought to the Harriet Lane Home and the following morning another bloody stool was passed; the first half of this stool was of a deep reddish-brown color, while the second half was light yellow, indicating that the bleeding had stopped abruptly.

Physical examination on admission showed a well developed but somewhat undernourished baby of 13 months who appeared to have lost some weight and looked a bit sallow; he was alert and not acutely ill. Eyes, nasopharynx, chest and abdomen were all normal.

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The hemoglobin was 55 per cent, the red blood cells were 4,300,000 and the white cells 9,600, with 67 per cent lymphocytes. The platelet count was 275,000. Clotting and bleeding times were normal. The urine showed a very faint trace of albumin on admission, none on later examination.

The child was immediately matched for transfusion in case of necessity, and exhaustive search was begun for the cause of his illness. The result of the tests and the condition found at operation are quoted from Dr. Guild's report to the family physician:

"Although various possibilities suggested themselves to us clinically, no positive evidence of any of them could be obtained on examination. The normal blood picture (apart from the anemia) with abundant platelets excluded bleeding on the basis of a blood dyscrasia. The absence of splenic enlargement ruled out bleeding from varices on the basis of a Banti's disease or splenic vein thrombosis. Stool culture showed no pathogenic bacteria and blood agglutinations against the typhoid-dysentery group of organisms were all negative, indicating that ulceration anywhere on the basis of any specific intestinal infection was unlikely. We could find no telangiectasis in the accessible mucous membranes to suggest that the condition of multiple hereditary telangiectases with lesions in the gastro-intestinal tract was present. Proctoscopic examination was also negative. Skin tests for allergy were negative to all foods, and, with regard to bacteria, there was slight sensitivity to the hemolytic streptococcus only. Some hemolytic streptococci were present in the throat on culture, although there was no acute inflammation of the throat while the baby was in the hospital.

"Of the remaining possibilities, there were three that we could not altogether exclude. Gastric or duodenal ulcer, though very rare in children, could have accounted for both vomiting of blood and the appearance of blood in the stools. An ulcerated Meckel's diverticulum or intestinal polyps could have accounted for the symptoms and for the blood that appeared per rectum, although neither (unless a polyp was also present in the stomach) could account for the blood that was vomited. The patient's mother stated that there had been some question as to whether the blood that was vomited might have resulted from the violence of the baby's retching. At any rate, we felt that these possibilities should not be dismissed too lightly. X-rays of the gastro-intestinal tract (G. I. series and barium enema) failed to provide any positive evidence for any of these, but we did not feel that negative evidence of this kind was conclusive. Other laboratory data, including intradermal tuberculin, blood Wassermann reaction, urine, chest x-ray, and so on, were all negative.

"At the time of the baby's arrival at the hospital he seemed in fairly good condition, and his hemoglobin was 55 per cent. A few hours after arrival he passed a large stool, the first half of which was of a deep reddish brown color (old blood), while the second half was of a light yellow, indicating that the bleeding had stopped abruptly. He was kept on a bland diet of buttermilk and cereal, so that any slight bleeding could quickly be detected by the color of the stool. There was no further bleeding during the next week and his hemoglobin rose to 70 per cent. At the end of the first week, however, following a day when he had seemed rather listless, he passed two large tarry stools and there was a concomitant fall in hemoglobin to 50 per cent. (This was four days after barium had been given by mouth for the G. I. series and before the barium enema or proctoscopic examination had been made, so it definitely had no relationship to any diagnostic procedures.) Again the bleeding stopped abruptly, as the two tarry stools were followed by normal ones within twenty-four hours. On one subsequent occasion there was a smaller tarry stool, with a final hemoglobin level of 45 per cent. No abdominal masses were felt at the time that the bleeding occurred, and there was no abdominal tenderness. The baby vomited only once and that was not at a time when he was bleeding nor was there any blood in the vomitus. During his stay on the ward, after he had become accustomed to being without his mother, he was playful and contented most of the time, although occasionally he had periods when he would draw his legs up under him and cry as if he were in pain.

"In view of the fact that, during this period of observation, there was continued evidence of bleeding, once sufficient to cause a drop in hemoglobin of 20 per cent, in a child who had had a previous bleeding episode five months before with intermittent ill health and occult bleeding at times since then, we felt that it was advisable to subject the baby to an exploratory operation, since his position with regard to hemorrhage was a precarious one and exhaustive clinical investigation had failed to clarify the situation. Meckel's diverticulum and polyps have been found in other cases in which clinical investigation has proven equally unenlightening.

"In preparation for the operation he was transfused on May 18 (without reaction) and the operation was performed on the morning of May 19. No lesion was found in the stomach or duodenum, there was no Meckel's diverticulum and there were no polyps. The only findings were some thickening of the wall of the descending colon and a tight band of adhesions extending across the transverse colon, with some enlarged mesenteric lymph nodes in the neighborhood. It seemed possible that this could have caused kinking or partial obstruction from time to time perhaps with temporary overdistention and rupture of blood vessels in the obstructed portion, although this was not a wholly satisfactory explanation for the baby's symptoms. It was hoped, however, that release of the adhesions would help the situation, and this was accomplished. The appendix was also removed, as it presented certain anatomical abnormalities that might give rise to difficulties later."

The baby stood the operation beautifully. On the afternoon of the day of operation, however, he had unexplained fever (106°), but took water well and did not vomit. The next day his temperature was lower (102°) in the morning, but rose to 107° at 8 p.m. During the afternoon he had seven fluid stools, none of which contained blood. High fever and watery diarrhea continued throughout the night, and the following day he went into sudden circulatory collapse and died, despite adequate amounts of fluid by continuous intravenous administration.

We were confronted then with two unexplained conditions, namely: intestinal bleeding and a fatal circulatory collapse with hyperpyrexia. The autopsy made quite clear the cause for both of these conditions. An infected cavity in the pancreas communicating with the pancreatic duct

was the obvious source of the bleeding into the gastro-intestinal tract, and extensive hemorrhages destroying both adrenals explained the collapse and death of the patient. Dr. Frank Kindell's description of these lesions is as follows:

"The pancreas appears to be normal as to size. Just distal to the head of the pancreas one finds a cavity 2x1 cm. This contains a small quantity of fluid, one or two little soft blackish red masses suggesting old clot, and a considerable quantity of yellowish-white gritty calcified particles. These are easily crushed. The wall of this space is yellowish. Outside it, there is an irregular zone of gray fibrous tissue in the surrounding pancreas and fine gray streaks suggesting scar. The duodenum is somewhat adherent to this area, but no connection between the cavity and the duodenum can be found and the mucosa over it appears perfectly normal. The cavity seems to lie definitely within the pancreas.

"The pancreatic duct is dissected out and the main duct is found to pass by the cavity. The duct is not distended and its wall is pale and smooth. As the main duct is followed outside the cavity it is found to come actually to the surface of the pancreas and there is some fibrous tissue around the duct suggesting that there has been loss of pancreatic tissue in this area. The opening of a good-sized branch very readily admits a probe and this passes directly into the cavity. The passage is only 1 mm. or 2 long, so that the cavity has ready communication with the main pancreatic duct. The duct opens into the duodenum just at the margin of the ampulla.

"Along one side of the wall of this cavity in the pancreas, there is a reddish mass of clot, slightly mottled, suggesting thrombus. This is about 8 mm. in diameter. No definite vessel is identified at the time of autopsy. Where a part of the clot is broken away in making a section, it shows a little red spot and a probe can be passed into this for several millimeters. This area will be dissected later.

"The cause of this lesion is not clear from the gross appearance, but its presence affords a reasonable explanation for the history of intermittent bleeding and abdominal pain. With the free communication between the cavity and the pancreatic duct, the erosion of vessels in the wall with hemorrhage could very well lead to hemorrhage into the intestine. Clots, if any, that formed in the pancreatic duct would no doubt be digested in the pancreatic juice. The presence of small calculi and of scarring around the cavity is evidence that it has been there for some time.

"It is surprising that there is so little gross change in the rest of the pancreas. No gross fat necroses are seen. As stated above, the pancreatic duct shows no gross lesions. The rest of the pancreas does not seem atrophied or scarred, although the lobules are bound together a little more tightly than usual.

"Adrenals. Both adrenals together weigh 10 gm. They are about equal in size. Externally they are uniform dark red. On section, one finds the cortex almost entirely converted to a reddish, rather opaque tissue suggesting hemorrhagic infarct. In some places the cortex does not seem to be entirely destroyed. In one adrenal the gray medulla can still be seen on section. Both renal and adrenal veins were opened before removing the adrenals and no large thrombi are found in them. On section, no small veins can be seen containing clots."

A search of our records for patients with either of these lesions failed to reveal any other instance of a similar pancreatic cavity. This lesion must be exceedingly rare. Extensive adrenal hemorrhages, however, have been found here in more than twenty autopsies. The lesion is certainly much more frequent than reports in the literature indicate. In infants and children there seem to be two distinct groups: first, hemorrhages in the newborn, and secondly, those associated with infections. The former appear a few days after delivery and are sometimes recognizable as bilateral palpable tumors (hematomata). The predominant clinical manifestation is a rapid respiratory rate, so rapid, in fact, that Goldzieher[1] has suggested the term, pseudopneumonia infantum. The cause of these extensive adrenal hemorrhages in the newborn has not been clearly established, but it is certain that infection does not play a role. Actually these hematomata may be only an exaggeration of a normal physiological process, for the central or androgenic zone of the adrenal of the embryo and of the newborn normally has extravasations of blood. These hemorrhages were thought to be due to trauma, but their existence during fetal life and in babies delivered by cesarean section precludes that explanation.

The second group of patients with hemorrhages in the adrenal glands are those in whom there is an overwhelming infection. In most of these cases thus far reported in the literature the invading organism was the meningococcus.[2] The infection is so fulminating that death occurs before meningeal signs appear. In some of these patients there are petechial hemorrhages in the skin and viscera simulating purpura fulminans. The bleeding into the adrenals, however, is more than a conglomeration of petechiae; it is extensive and destructive of both the medulla and the cortex. The name Waterhouse-Friderichsen syndrome has been proposed for these cases of severe meningococcemia associated with fresh and extensive hemorrhagic destruction of the adrenals. There have been two such cases at the Harriet Lane Home in which the diagnosis was established at autopsy. There has not been

any case of epidemic meningitis in which the complication was recognized clinically.

The contention that most of the cases of Waterhouse-Friderichsen syndrome are due to meningococci is not supported by the findings in the eleven cases in the Harriet Lane Home. The records of this branch of Johns Hopkins Hospital show that severe adrenal hemorrhages, associated with hyperpyrexia and circulatory collapse, have been found at autopsy in three children dying of staphylococcus septicemia, in one patient with erysipelas, in one with Streptococcus viridans endocarditis, in one with streptococcus infection complicating a burn, in one with bronchopneumonia, and in one with unexplained fever. The last patient was a 4 months old baby who was admitted because of an extremely acute nutritional disorder and lobular pneumonia. There was an intractable disturbance in water absorption despite vigorous administration of parenteral fluids. The autopsy showed definite thrombi in the adrenal veins, and the pathologist thought that these looked older than the adrenal hemorrhages.

In none of the foregoing cases was the diagnosis of severe adrenal hemorrhage suspected during life. Every one of the patients had high fever (104 to 107°) and died in a short time after signs of circulatory collapse appeared. Not all of the patients had positive blood cultures, but all had unmistakable evidences of severe infection.

The mechanism causing destructive hemorrhages from the adrenal vessels is not an obvious one. Venous thrombosis has been suspected as the initial change. It can be pointed out, however, that in only one of the eleven Harriet Lane Home cases was thrombosis seen at autopsy. Furthermore, in the case reported in full in the earlier part of this paper, Dr. Kindell examined the adrenal veins with extreme care before removing the glands from the body and found them patent. A second possible explanation is that of Kraus,[3] who suggested that the adrenal veins have an unusually thick muscular coat which becomes spastic and thereby favors extravasation into the parenchyma of the gland. The German literature has many articles debating this theory, but it is not one that is amenable to experimental study or clinical proof. Still another explanation of the adrenal hemorrhage is that some bacterial toxins cause an increased permeability and rupture of the venous walls. Recently McClean[4] has shown that alteration in cell permeability is produced by certain bacterial toxins. The question arises why the adrenal veins should be more vulnerable than other veins. This vulnerability may be due to some biological action of the glands, but more than likely is due to the relative increased blood flow through these veins. In this connection it is interesting to note that many patients with adrenal hemorrhages show widespread petechiae elsewhere in the body. Furthermore, during the first three years of life the central or androgenic zone is undergoing involution and has a far richer blood supply than the medulla of the mature gland.

Finally, one is confronted with the practical aspects of adrenal hemorrhage, which consist of clinical recognition and effectual treatment of the condition. At present there are no tests which point unmistakably to the diagnosis, and the best that we can do is to be on the alert when unexplained hyperpyrexia occurs in cases of infection due to toxin-producing bacteria. The treatment of such cases when promptly diagnosed would seem to call for operative interference. Corcoran and Straus[5] reported in 1924 that they had recognized this lesion clinically and had successfully evacuated the clots from the adrenal glands. Probably many lives will be saved if operative treatment is combined with the intravenous injection of a potent cortical hormone, such as that described by Grollman.[6] Our experience in the laboratory with the commercial cortical products now on sale has destroyed our confidence in their potency, but this situation does not preclude the possibility that potent extracts will become available to the practitioner in the future.

Our contact with the patient whose illness is described in the first part of this paper not only provoked a survey of similar cases in this hospital, but has served as a stimulus to experimental study of the problem of acute adrenal hemorrhage. We are trying to learn how the bacteria or their toxins damage the adrenal veins and why these veins are more vulnerable than those of other organs.

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THE PRESENT STATUS OF FLEXIBLE TUBE GASTROSCOPY*

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Flexible tube gastroscopy, having passed from a medical stunt to a safe, simple procedure, I should like to present a summary of its status in a clinic where it has been used more or less routinely for fifteen months.

JUSTIFICATION AS A ROUTINE PROCEDURE

We believe that examination by the flexible gastroscope should be offered to every patient with a gastric complaint, without definite contraindication. These contraindications are: first, general conditions severe enough to render any instrumentation unwise; and, second, any lesion of the esophagus, or lesion outside of the esophagus which encroaches on its lumen.

The procedure is safe enough for routine use. Out of thousands of examinations there have been only five known accidents to date. These were perforations of the gastric wall. Three were sutured and two were let alone; all made uneventful recoveries. These perforations all occurred with the sponge tip, which has now been abandoned. There have been no known accidents when the rubber finger tip was used. There have been no known perforations of the esophagus. In addition, the ease of detecting resistance to the passage of the gastroscope down the esophagus, and the slight pressure required for insertion when using the rubber finger, have convinced me that in skilled hands, using reasonable care, the danger is only slightly greater than when using the large stomach tube.

It is not a major procedure to the patient. The passage of the gastroscope and the manipulation for a considerable period of time does not seem particularly harrowing. Patients generally consider the procedure as slight and apparently have little discomfort. With rare exceptions, they will consent to repeated examinations with no more animosity than toward repetition of the x-ray.

As far as the accuracy of observation is concerned, the vision obtained through the instrument is perfect.

A major question is the completeness of the examination. There is some doubt, with the instrument as it is at present, that all of the stomach may be seen.

First, it has been claimed that an area of varying size, situated on the lesser curvature of the antrum, close to the angle, is a blind area. I believe, in many cases, that this area can be seen with the aid of peristalsis, which brings it down into view. Second, there is an area on the greater curvature, ahead of the instrument, that is supposed to be blind. Although I have not adequate proof as yet, I am certain from observations that this area can be seen almost without exception from above, using varying degrees of inflation. The most difficult region of all to visualize is the posterior wall near the lesser curvature at the entrance of the esophagus to the stomach. This area can be visualized frequently just as the instrument is withdrawn. Manipulation of the stomach by manual pressure on the anterior abdominal wall aids in obtaining a view of the pylorus and difficult areas in the lower portions of the stomach. Dr. Chevalier Jackson has frequently mentioned this point in his work with the open tube gastroscope.

Therefore, I do not believe there are any true blind spots. Certainly one cannot hope for a complete examination in every case and in a few cases it is impossible, as, for instance, when there is a chronic, benign lesion of the pylorus. In this case adhesions and spasm frequently pull the pylorus and distal portion of the antrum from view. In the great majority of cases complete inspection is possible, although one may have to repeat the examination and vary the technic slightly.

An important point to be realized is that one can become so well oriented that when an area is not seen he is aware just which area was not visualized. This means that a negative diagnosis is justifiable.

It is certain that the instrument discovers information concerning common ailments that can be obtained in no other way so easily.

These points have convinced me that flexible tube gastroscopy fulfills the requirements of a routine procedure.

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GASTRITIS

The greatest single service that the flexible gastroscope has rendered is that it has revived the question of chronic inflammation of the stomach. Although almost all phases of the question are still in the experimental stage, and hot dispute rages around almost every point, some things are apparent to everyone who has had any experience with the procedure.

When one gastroscopes a series of patients with various digestive symptoms, but with x-ray negative stomachs, it becomes apparent very early that these patients can be separated into groups on the appearance of the gastric mucosa. In a certain percentage, the mucosa of the stomach is normal. This is to say, if seen on any other mucosal surface, it would be considered normal. A certain percentage of these stomachs show mucosas that, if seen anywhere else, would be considered abnormal. The remainder will show various gradations between the two extremes, so that one is hard put to it to say to which group these belong.

It is further evident that these mucosas which appear abnormal fall naturally into three distinct groups. In one group of cases the mucous membrane appears thin, bluish to bluish gray in color, in contradistinction to the orange red of the mucosas that we call normal. The rugae are small, widely spaced and tend to disappear with inflation. Further evidence of the thinness of the mucosa is that usually bluish vessels resembling the veins in the retina are seen running across the field. This change we refer to as atrophy.

In marked contrast to these are the mucosas which appear thick, and in places have a granular appearance. The color is a deeper red, sometimes a beefy red and loses the yellow element. The rugae throughout their length may be thickened and contain fusiform swellings. There may be nodules on the mucosa, occasionally on the crest of the rugae, but usually in the valley. The mucosa bears some resemblance to the pharyngeal mucosas when chronically inflamed and thickened. We refer to these stomachs as hypertrophic.

The third group shows no thickening or thinning of the mucous membrane, the normal translucent appearance is not lost, no nodules or granules are seen, but the mucosa appears reddened, particularly on the crests. There are small localized reddened areas and hemorrhagic areas resembling the thin flat hemorrhages of the ocular sclera. Small erosions may be present, and occasionally bleeding points. The main point is that the changes seen in these stomachs appear to be surface changes. We refer to this type as superficial.

We consider these changes as presumptive evidence of disease and tentatively apply the label gastritis. We must grant at the moment that this presumption rests chiefly on analogy. We find that almost all healthy, symptomless individuals have gastric mucosas of the normal class. We find that in patients with digestive complaints, but without other evidence of disease, about 40 per cent show abnormal mucosas.

In my own series from the Johns Hopkins Hospital Gastro-Intestinal Clinic, which is as yet inadequate for final conclusions, 38 per cent of these cases show abnormal changes, 21 per cent are borderline, and 41 per cent must be classified as normal.

In cases where definite trauma or disturbance of gastric integrity has occurred, as in postoperative stomachs, these inflammatory changes are almost always present. Over four out of five in my series have shown marked inflammation, either general or localized around the stoma.

In duodenal ulcer the incidence of these gastric inflammatory changes increases greatly.

Further analogy can be found in the atrophic group, for we find the same appearance, though usually more marked, in the gastric mucosa of pernicious anemia cases where atrophy is known to occur.

I believe these are true pathologic changes, and that they can and do cause symptoms, and that normal physiology is interrupted, the degree of subjective discomfort depending, as in other mucosal disease, on the severity and extent of the process, and possibly its location.

However debatable most of these points may be, one important fact stands out. These abnormal appearing stomachs bleed, varying from slight to large amounts, and these bleeding areas can be located through the gastroscope. No opinion based on occult or gross hemorrhage is worth anything until the gastroscopic findings are known.

These three types comprise the classification of Schindler. There have been other attempts to subdivide these groups into many, but I do not believe them justified at this time. It seems to me that the danger is in attempting any fine classification. These abnormal changes fall naturally into these three groups, but even the

question whether they are merely different stages of the same process or whether they are disease at all is still unproven.

The superficial changes may be seen in stomachs that show atrophy or hypertrophy in other areas. It has been denied that atrophy and hypertrophy may occur in the same stomach, but I have seen at least one case of localized atrophy in which the remainder of the stomach certainly looked hypertrophic, although no nodules were seen. Hemorrhages, erosions, color changes, and so on, seem to involve all types so that when we get to the smaller classifications such as Moutier has attempted, overlapping produces a hopeless jumble.

Therefore I believe that in the present state of our knowledge, for all practical purposes, this simple natural classification should be used, bearing in mind that we may be speaking merely of one process, chronic gastritis, and remembering for research alone that there is some indication that a further classification may be possible. For instance, I have repeatedly observed reddening and an apparent granularity rather sharply limited to the pre-antral smooth zone, and there are other criteria that may serve as a basis for departure from the fundamental classification, such as multiple erosions, hemorrhages, verrucous nodules, or variations based on the three types of Schindler. Most of these problems only time will solve.

ULCER AND NEOPLASM

My own experience has been limited with both of these. However, there are a few things that I can say.

A benign ulcer has a characteristic appearance. The floor of the ulcer is a grayish or yellowish white, the edge is sharp and very sharply defined. In all that I have seen the surrounding mucosa was reddened and edematous, and some of the surrounding folds were thickened. The diagnosis is easy. The ulcer fairly leaps into view, and it seems impossible to overlook any ulcer that might be in the field of vision.

A malignant ulcer has an altogether different appearance. The floor is darker and has a purplish brown color. The edges are ragged, the infiltration into the surrounding tissue is obvious in yellowish white ridges, and the surrounding mucosa may or may not be inflamed.

The non-ulcerating carcinomata that I have seen appear as yellowish white elevations of the mucosa, with irregular infiltrating edges. It is quite evident that neither of these resembles the benign ulcer.

I have had no experience with benign neoplasms of the gastric wall.

The truth of what the more experienced gastroscopists have contended is obvious to me even with limited experience, namely: First, that there is no other method that can equal the gastroscope in differentiating benign peptic ulcer from carcinoma.

Second, the gastroscope offers a simple method for the early diagnosis of gastric cancer. I have no statistics, but it is obvious from the appearances that carcinoma should be recognized however early one might view it. Dr. Schindler says that he has never missed a carcinoma of the stomach, although he admits having missed a small percentage of ulcers. One of the difficulties in benign ulcer is that adhesions and spasm frequently pull the pylorus and part of the antrum from view. In a malignant lesion this does not seem to occur.

The present status of the procedure in ulcer seems to be about on a par with x-ray in discovering the ulcer, although it must be borne in mind that the same ulcer is not likely to be missed by both procedures. The gastroscope's particular value seems to lie in the more certain differentiation from malignancy, the state of the ulcer, evidences of healing or spread, and the reaction in the surrounding tissue. This information may well change some of our concepts of the handling of ulcer.

In gastric carcinoma, the gastroscope bears promise of superiority to any other method in detecting early lesions. It can probably give more accurate information as to the extent than any other method.

Another field in which flexible tube gastroscopy is unequal is in determining the state of a gastro-enterostomy. Not only the question of function may be decided, but the presence or absence of ulcer in the stoma. While it must be said that a posterior gastro-enterostomy, with severe inflammation of the surrounding area offers a considerable technical problem at times, still with manipulation and patience the stoma and occasionally the jejunum may be seen. A marginal ulcer in the stoma is clearly visible.

RELATION TO THE X-RAY

The gastroscope is not to be considered as an alternate to x-ray examination of the stom-

ach. It is an additional examination. Both contribute information; both present the same information from different aspects; each has its own sphere which the other does not enter. One is a silhouette, the other direct vision.

In the first place, there are certain fundamental differences, aside from the pathologic differentiation. The stomach inflated with air and viewed directly is much more complex than the barium filled silhouette of the x-ray.

Certain structures which are not presenting features in x-ray, if they are seen at all, are outstanding through the gastroscope. First and foremost are the rugae. These are thick, closely spaced, and tortuous, on the posterior wall, and are thin, parallel and widely spaced on the anterior wall. I have never seen an x-ray film which shows these as we see them. Occasionally they are partly delineated, but never clearly. The large, longitudinal folds seem in almost every fluoroscopy and sometimes referred to as rugae, using their thickness or thinness as a basis of a diagnosis of gastritis, are not rugae. I do not know what they are. Perhaps, as Dr. Schindler suggests, they are folds caused by contraction of the muscularis. I have noticed thick folds like this in the slightly inflated stomach. Again, the x-ray usually shows rugae or folds in the antrum, and they are very infrequent through the gastroscope. Viewed through the gastroscope, the lesser curvature is usually free from rugae, and is seen as a broad, smooth, shallow groove. The musculus sphincter antri, a thick ropy, barrier, forming the anterior-inferior border of the antral entrance is not seen by x-ray.

Peristalsis of the body can be observed satisfactorily only by fluoroscopy, although it is occasionally observed through the gastroscope. Peristalsis can be observed equally well in the antrum by both methods, slightly different information being given by each.

I have already discussed the two methods in ulcer and carcinoma.

In determining the abnormal mucosal changes discussed under gastritis, there is no comparison. I am convinced that these lesions, except in very marked cases, cannot be demonstrated by present x-ray methods, and I do not believe that the small lesions can ever be demonstrated by silhouette. It is self-evident that the small hemorrhages, the reddened areas, the edema of the membrane can never be shown in this manner.

The great advantage of the x-ray is that the stomach may be observed in relation to the duodenum and pyloric canal.

Gastro-Photography.—One more point should be mentioned because of present misconception. This procedure has no relation to gastro-photography. No photograph has been made through this instrument. Further, no photograph which does not record natural color, such as those made through the rigid instrument, will ever have any value.

SUMMARY OF THE PRESENT VALUE OF GASTROSCOPY IN EVERY-DAY PRACTICE

Taking for granted that each examination may not be complete and that in some cases the entire stomach may never be seen, a gastroenterologist may expect the following benefits from the use of the flexible gastroscope.

First and foremost, given a patient with a gastric complaint, we may definitely decide whether or not a lesion is present.

Second, we can determine the extent and nature of that lesion.

For instance, one has a safe, easy means of determining whether a patient in the cancer age with gastric symptoms has cancer or not, without having to lose valuable time waiting until the diagnosis is obvious and the cancer probably inoperable. If cancer is present one can reveal to the surgeon the extent of the operation to be undertaken.

Third, if the lesion is ulcer one can more easily follow the course by means of the gastroscope than by relying on the x-ray niche, which, after all, is not the ulcer.

Fourth, one may directly inspect a gastroenterostomy stoma for ulcer.

Fifth, one may subdivide his dyspepsia cases into those with apparent organic mucosal changes and those without. Although the treatment may be identical at the present time, we have reason to believe that the usual methods of treatment are not sufficient. In addition, there is some evidence that these changes may be fertile soil for more serious disease.

DISCUSSION (*Abstract*)

Dr. Rudolph F. Schindler, Chicago, Ill.—The rediscovery of the so-called minor diseases of the stomach, which only too often are major ones or which are the soil for major ones, has been of the greatest importance. I am happy that Dr. Borland, according to his observations, has adopted my classification of chronic gastritis, which

is the only one in which the gastroscopic appearance corresponds with clinical entities. However, I like to choose another subject to give you an idea of the possible future of gastroscopy (which now is a safe and not uncomfortable procedure).

We have a systematic successful fight against cancer of the breast and of the uterus; but we have no systematic fight against the more frequent cancer of the stomach. Why not? Boas' opinion is quoted much too often that the beginning of stomach cancer makes no symptoms and that it is inoperable as soon as there are symptoms. This opinion is correct in only a very few of our cases. Most of them have a history of slight stomach distress, especially lack of appetite for months and years. The true reason why we get few operable cases of stomach cancer is that neither the public opinion nor the physicians as a whole nor even the gastroenterologists have any confidence in surgery in gastric carcinoma. They believe that almost inevitably every patient dies from stomach cancer. In my experience, based on over 3,000 gastroscopies, they are entirely wrong. I know of four cases who outlived the operation for more than ten years, a large number who lived longer than 3 to 5 years. However, the fault is that, except in hopelessly advanced cases, almost every tumor is operated upon to give the patient a last chance. I myself handled these cases in exactly the same way before the invention of the flexible gastroscope. This instrument, however, permits us to decide with one glance upon the operability of a stomach cancer so far as the involvement of the gastric wall itself is concerned. It must be emphasized that gastroscopically usually even the smallest beginning tumor can be easily seen, as Dr. Borland has pointed out.

We may subdivide gastric carcinoma grossly in four groups:

(1) The polypoid tumors, broad based, prominent, usually not ulcerated, sharply defined. They can be operated upon wherever their site may be.

(2) The ulcerations surrounded by a high, sharply limited wall. This type is frequent and gives an excellent operative prognosis. A "meniscus" carcinoma seemed to me to be inoperable because of the high seat, but gastroscopy showed a sharply demarcated wall. Therefore subtotal resection was performed in 1930, and when I left Munich in 1934 the patient still lived and felt well.

(3) Ulcerated tumors which are only partly sharply defined. In these operations can be performed successfully only if the infiltration proceeds toward the pyloric region. The x-ray picture of such a tumor may look completely benign with smooth converging folds. In the gastroscopic picture the ulcer was filled with clotted blood, a gray ridge was seen. That does not happen in benign ulcers. I diagnosed immediately beginning carcinoma. After three weeks the ulcer was shallower, also in the x-ray picture, but now there was an infiltration with formation of nodes toward the pylorus. This is a type 3 operable carcinoma. Operation proved the correctness of this early diagnosis.

(4) Carcinomas of type 4, which are diffusely infiltrating, are always inoperable, even if they are very small. However, x-ray relief cannot decide their extent. An ulcer by x-ray may look carcinomatous, but gastroscopically one may find a totally unsuspected extensive carcinomatous necrosis and infiltration which extends upward to the cardia.

Operability of a gastric carcinoma, aside from metastases, can be decided by gastroscopy, and only by gastroscopy. If we could operate only upon our operable cases the results would improve suddenly, and we should regain the confidence of the public opinion and of the physicians. A campaign could be started to examine thoroughly every patient suffering from slight lack of appetite, loss of weight, and abdominal symptoms, by x-ray relief and by gastroscopy.

The question still remains: what shall we do with our inoperable cases? I have the impression that the diffusely infiltrating tumors respond best to high voltage therapy after Coutard. I will not quote any cases; this question is not settled at all; also here gastroscopy observation of the results is indispensable.

After these considerations we can make a plan for a systematic fight against stomach cancer, with the following seven points:

(1) Exploratory laparotomy must be eliminated completely by gastroscopic determination of the type and operability of the carcinoma.

(2) In each inoperable case of type three or four, energetic high voltage x-ray therapy should be tried in order to come to definite conclusions concerning the value of this type of therapy.

(3) If after the discarding of exploratory laparotomies the confidence of physicians and public opinion in surgery of stomach cancer has been reestablished, a widespread campaign concerning early diagnosis should be brought about. Both practitioners and laymen should learn that immediate thorough examination of the stomach, consisting of both x-ray and gastroscopy, becomes necessary and profitable when in people over 35 years, unexplained lack of appetite, loss of weight and slight abdominal symptoms are observed.

(4) A sufficient number of physicians have to be trained thoroughly in both the x-ray relief method and gastroscopy.

(5) Each patient in whom atrophic gastritis or benign epithelial tumor, in my opinion the forerunners of carcinoma, have been found previously, must be examined twice a year by gastroscopy and x-ray relief method.

(6) Each patient suffering from superficial gastritis has to be treated most carefully under gastroscopic observation in order to prevent development of atrophic gastritis which may be the soil for the origin of cancer.

(7) Each patient over 35 years in whom ulcer of the lesser curvature has been discovered by x-ray must be gastroscoped immediately.

The points (1) gastroscopic determination of the operability, and (7) gastroscopic differentiation of benign and malignant ulcer, seem to be the most important ones in the beginning.

If we consider that the cancer problem is only a small part of the usefulness of gastroscopy, then we shall understand why gastroscopy has suddenly assumed an important position among the daily methods of examination. It is the great merit of Dr. Borland to have shown you this development and this position in detail.

Dr. E. B. Freeman, Baltimore, Md.—I wish to discuss briefly three unusual cases. Two of the three had been diagnosed carcinoma of the stomach, and one had been diagnosed gastric hemorrhage of undetermined origin. In the first case, an x-ray diagnosis of carcinoma of the body of the stomach had been made. The

gastroscopic examination failed to show any evidence of malignancy. This was one of our early cases, and we thought that our inability to find the lesion was due to lack of experience. This patient was operated upon and no evidence of malignancy was found. In the second case, an x-ray diagnosis of malignancy of the stomach involving the prepyloric region had been made. Gastroscopic examination showed the malignancy to be very much more extensive. Cancerous nodules were scattered throughout the body of the stomach to within a short distance of the cardia. This patient was also operated upon, and nearly all of the stomach was found to be involved in the cancerous process. These two cases suggest that gastroscopy is of value not only in helping to determine the presence of carcinoma, but also in determining the extent to which the stomach is involved, and whether or not anything is likely to be gained by operative treatment.

In the third case, a diagnosis of gastric hemorrhage of undetermined origin had been made. There was a history of repeated attacks of vomiting of large quantities of blood, occurring at long and irregular intervals for several years. Physical examination showed enlargement of both liver and spleen. X-ray study failed to reveal a lesion in the stomach or duodenum. In view of the fact that a lesion was not demonstrated in the stomach by x-ray study, it was thought that the hemorrhage was probably associated with cirrhosis of the liver. An esophagoscopic examination was made, which showed that the hemorrhage was coming from esophageal varices. In all cases of gastric hemorrhage where cirrhosis of the liver is even suspected, an esophagoscopic examination should be done to determine whether or not the hemorrhage is coming from the esophagus. A gastroscopic examination should never be made if esophagoscopy reveals the presence of esophageal varices.

Dr. Porter P. Vinson, Richmond, Va.—We are greatly indebted to Dr. Borland for his splendid presentation of the value of direct visualization of the stomach with a flexible gastroscope and to Dr. Schindler for his illuminating discussion.

One might be inclined to doubt the accuracy of the drawings which were shown and to consider the colors more vivid than they would appear at gastroscopic examination. I should like to emphasize the fact that they are true representations of lesions as seen with the Schindler gastroscope. Further modifications and improvements of the instrument may be necessary before it is perfected, but at present it is sufficiently valuable to warrant its employment in the examination of almost all lesions of the stomach. In skilled hands the examination can be made with a minimal amount of discomfort and risk to the patient.

Dr. Borland (closing).—This procedure should be made a routine in every gastro-intestinal clinic. No patient can be considered as having had a complete examination without gastroscopic inspection. Of course the method is new. The pathology has not been thoroughly worked out and even fundamental points remain unsolved. Much work remains to be done on such things as the function of the musculus sphincter which in conjunction with the angulus seems to act as a barrier to the antrum and appears in some way to modify peristalsis. It is possible that this may have some bearing on surgical procedure, such as location of a gastro-enterostomy. The fact that gastritic changes are frequently localized to definite zones makes correlation of physiological, histological and gastroscopic boundaries an important problem. Such things as the effect of drugs, effect of diets or of heat and cold on the gastric mucosa need to be evaluated. It seems to me that the various methods of ulcer treatment need to be reevaluated by following the ulcer through the gastroscope. There, of course, are only a few of the problems that must be solved in the immediate future.

CARCINOMA OF THE GALLBLADDER*

STUDIES OF 24 CASES IN GEORGIA

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and
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This subject has been so thoroughly discussed during the past year by Illingworth[1] and Boyce and McFetridge,[2] with whom we most heartily concur, that we shall confine our remarks to an analysis of cases seen in the University Hospital at Augusta and the emphasis of several points. These are added to the growing list of reported cases with the forlorn hope that ultimately some clue suggestive of cancer at a much earlier stage may be found. Certainly we shall make no progress in the treatment until we learn to recognize the disease early enough for removal, or deal more energetically with prophylactic measures. At the present time we must concentrate upon the latter.

Age, Sex, Race.—Of 24 cases in the University Hospital the youngest was 26 years of age, the oldest 78 years; 23 were 40 years or over, 19 were more than 50 years old. Eight were men and 16 women, a larger ratio of males than in most previous reports. There were 20 whites and 4 colored (2 men and 2 women). Considering the rarity of cholecystitis among Negroes, this is a rather unexpected finding. In checking up our gallbladder cases two years ago, we did not find a single instance of proven cholecystitis in a Negro patient, and I know of none during the past two years. This is a marked variance with reports from the Charity Hospital in New Orleans, where there is a ratio of 1 Negro to 6 whites. A recent inquiry in Savannah, Georgia, elicited the fact that in a large Negro hospital there only 4 or 5 cases had been operated upon in the last 25 years. The 26-year-old patient

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was a Negro woman who had complained for two years of stomach trouble and pains after eating, the pain localized in the right upper quadrant of the abdomen. There had never been either nausea or vomiting. She was jaundiced, had bile in the urine and stools and a smooth mass in the region of the gallbladder which moved with respiration and was not tender. The liver was palpable on either side of the mass. The temperature was normal and white blood count was 16,000. She had lost 15 pounds in weight. X-ray was negative for stones. At operation an inoperable cancer was found with extension into the liver and lymph nodes. No stones were palpable. Diagnosis was confirmed by biopsy. Our next youngest patient, 40 years old, was also a Negro woman with a similar history over a period of 18 months and similar physical, laboratory, operative and autopsy findings.

Etiology.—The prevailing opinion is that cancer develops upon a chronic cholecystitis and the presence of stones probably increases this tendency. A papillomatous gallbladder may be more susceptible to this irritant influence, but this is by no means proven.

Pathology.—Our classification is similar to that clearly defined in Illingworth's paper, which recognizes four principal types.

(1) The scirrhous carcinoma, by far the commonest, appears grossly as a diffusely thickened and indurated gallbladder which frequently may be mistaken, in its early stages, for chronic cholecystitis.

(2) The papillary carcinoma manifests itself by solitary well defined pedunculated tumors or by course villous and often fungating growths. In the villous type occlusion of the cystic duct not infrequently occurs early, causing marked distention of the gallbladder simulating an empyema. A grave error is made when cholecystostomy is performed in such cases as our study reveals. When whitish colorless bile is obtained on aspirating a distended gallbladder the possibility of malignancy should at once come to mind.

(3) The colloid type of carcinoma is characterized by its bulkiness and the soft jelly-like appearance of the growth. This type could probably be subgrouped in Group 1. It is merely an adenocarcinoma in which the secretory activity of mucin by the glandular epithelium is not only well preserved, but greatly exaggerated and perverted.

(4) The squamous cell carcinoma of the gallbladder, though said to be quite common by Oberling, has been seen but once by us. It is, however, a remarkable example of metaplasia. Grossly it is difficult to differentiate this from the scirrhous type.

Of especial significance in connection with the histopathologic study of our series of cases is the observation that the staining reaction of the epithelial cells, particularly in the scirrhous group, divides them into two main classes. The microscopic structure of a scirrhous tumor of the gallbladder is that of an adenocarcinoma. The acini or tubules are lined either by dark, well-stained columnar cells or by clear, large, poorly stained pale columnar or cuboidal epithelium. The morphological differentiation of these tumors into those with dark or pale stained cells is of importance when their relative malignancy is taken into account. The grading of gallbladder carcinoma is a procedure from which naught may be gained and this makes the attempt at prognostication most difficult and unreliable.

In some cases the appearance of cells and formation of acini are not greatly different from the normal, yet early metastases may be present. Mature looking glandular acini are frequently seen in the metastatic nodules as well as in the primary growth which, on examination, did not differ greatly from the deeply placed tubules seen in the crypts of Luska in the healthy gallbladder or the acini that penetrate the muscle coats in chronic cholecystitis and are known as Aschoff sinuses. However, we have noted that the carcinomas made up for the greater part of the large, clear pale stained cells grow more slowly, giving a longer past history of the disease, and survive longer after operation than those in the group composed of dark stained columnar cells regardless of their grading.

Symptoms and Signs.—Jaundice was present in 13, absent in 11 cases. Pain was a more constant symptom, occurring in 18 (of which 12 were proven to have calculi). Pain dates back longer in the histories than any other symptom and became progressively more pronounced from a dull aching to a constant boring as the disease developed, different from attacks of colic, and was the chief factor in driving the patient to the doctor. Calculi were proven in 14, none was found in 9 and their existence was undetermined in 1. Bile in the urine was found in 8 patients, all of whom had jaundice, but it is interesting to note that 5 jaundiced cases did not show bile in the urine. Seven had bile in the stools, 4 had none, and in 13 it was not mentioned in

either history or laboratory reports. Three gave a history of typhoid, 2 of malaria, 4 gave no history of either and in 14 neither disease was mentioned. Seven had had previous similar attacks. The usual biliary complaint of "indigestion" or dyspepsia was noted in only 12, and of these, 6 complained of flatulence or "gas." Fever was present in only 7 and 1 complained of itching at the time of admission, 1 gave a history of itching six months previously, but not at the time of admission. Eleven had lost weight, 3 of these as much as 50 pounds, and in these very extensive invasion was found at operation. Four had no loss of weight; in the remainder it was not mentioned. A palpable tumor was found in 13, palpable liver in 12; of these 7 had palpable tumor and liver, 5 had palpable liver but not tumor, 6 had palpable tumor but not liver, 18 had either palpable tumor or liver. Wassermanns were negative in 13, not recorded in the others. Bleeding time except one was not increased in any recorded case. Blood sugar reports were secured in 7 cases: 2 were high, 2 low and 3 normal. White blood count varied from normal to 16,000.

Duration of Symptoms.—Eight patients could trace their histories back one year or longer, one of these over a period of 20 years, which certainly must have signified an old cholecystitis, though she was only 57 years of age. Six had symptoms of from six months to one year's duration, 6 of six months' or less, and 4 unknown. X-ray studies of 15 cases showed no dye in 9 in whom visualization was attempted, but 1 of these showed a large calculus. In 3 others, stones were shown by flat plates. Gastro-intestinal x-ray studies and barium enemas in several proved the tumors to be extrinsic, 2 showing some distortion of the pylorus by pressure.

Previous Hospitalization.—Five had been in the hospital for similar complaints within the preceding year. Two of these had cholecystostomies with persistent sinus, 1 had a second cholecystostomy for the sinus six months after the first and on the last admission had a cholecystectomy, which revealed the cancer. One after some time began to drain minute plugs of tissue which microscopically proved to be cancer.

Preoperative Diagnosis and Operation. — In only 7 was cancer of the gallbladder or pancreas suspected; the remainder were diagnosed as gallbladder disease except that one was thought to be peptic ulcer. In 5 cholecystectomy was done, including the case mentioned above, cholecystostomy in 4, choledocho-duodenostomy in 1; the remainder (12) had exploration with biopsy in several and closure. Two were discovered at autopsy. Nineteen had positive laboratory diagnoses from operative specimens, including biopsies and autopsies. Five had only clinical diagnosis from operative findings. Fourteen showed extension to the liver or nodes or both. Three showed other tumors, 1 cancer of the face, 1 lipoma of the flank, 1 multiple adenomata of the thyroid and papilloma of the ear and the scrotum.

End Results.—All are dead, except that 1 living after five years and apparently well, and 1 explored two months ago revealed a densely indurated gallbladder with very extensive invasion of the nodes and liver and is very rapidly declining. She will probably not last the year out. (This patient died rather suddenly four days ago.)

DISCUSSION

Several of our cases emphasize the condemnation of cholecystostomy and argue for cholecystectomy. We must cure cancer of the gallbladder when it is an accidental finding, not when we have been able to diagnose or even suspect its presence. From this study two salient points appear: (1) a thickened gallbladder with enlarged lymph nodes suggests malignancy and should not be subjected to drainage only, but should be removed; (2) the aspiration of "white bile" from a thickened distended gallbladder with little or no fever, a normal white blood count, with or without jaundice is very suggestive. Fever may indicate a secondary cholangeitis or liver degeneration. Persistent pain with jaundice is quite suggestive of malignancy. An interesting though unexplainable observation by one of us (Greenblatt) is that the knee jerks were absent (Wassermanns negative) in several in whom the pancreas was secondarily involved.

That we can save only 1 in 24, or 4 per cent, is a terrific indictment against us. We answer, they came too late. But why? Because our clinicians are not sending patients with gallbladder disease to the surgeon as they should. Again why? Because both our mortality and our morbidity have been too high. Another why? Because we have not made sufficient use of laboratory studies, cholecystography, liver functional tests, blood chemical studies, including sugar, nonprotein nitrogen, chlorides, calcium, uric acid and others to evaluate the risk properly and to correct errors in these converting a bad risk into a good risk before we rush into operation. There

is no excuse for a 5 to 10 per cent mortality, as has been well shown by Graham in reducing his to less than 1 per cent, and others have done likewise. Of course the clinician was hesitant about referring the cases. As to morbidity, again we and the clinician have failed in that though the operation was a success (technically) the patient continued to suffer very much as he had. We failed to appreciate the fact that gallbladder disease is not a primary disease, but an end result of years of abuse of hygiene, of diet, of infections and so on and that until these are corrected the same abuse will be heaped upon the liver and the patient will continue to suffer. Remove the causative factors leading up to the production of the disease and our morbidity will improve. With mortality and morbidity lowered to the realm of reason we can expect the cooperation of the clinician in removing sites of chronic irritation, one of the chief factors in the development of cancer. Thus prophylaxis.

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DISCUSSION (*Abstract*)

Dr. Frank S. Lynn, Baltimore, Md.—The incidence of carcinoma of the gallbladder is higher than we usually think. Most carcinomata of the gallbladder are associated with gallstones, so much so that gallstones, in the majority of cases, are thought to be the etiological factor, bringing us back to our old theory that cancer development is due to irritation. It is also a noteworthy fact that most carcinomata of the gallbladder occur in women, and this also is in line with the belief that gallstones precede the development of cancer. Most authorities draw attention to the fact that carcinoma of the gallbladder is more common in women and carcinoma of the ducts more common in men.

Rolleston, in 1905, gave the incidence of this condition as 5.1 per cent; Deaver, in 1927, gave it as 1.5 per cent; Judd, in 1932, gave 1.4 per cent; so you see in a period of about thirty years the incidence has diminished from 5.1 to 1.4 per cent. This decrease may be explained as due to the more frequent removal of gallbladders, and to the prompter submission of patients suffering with gallbladder disease to operation. There do not seem to be any pathognomonic symptoms connected with carcinoma of the gallbladder; all patients seem to suffer with chronic gallbladder disease. If and when the neoplasm becomes palpable the case is usually inoperable. While it is thought by many that irritation due to gallstones is the commonest cause of this condition, we should not use this fact alone to influence our patients to be operated upon. To quote Erdman, "In using such an argument, one must be certain that one's operative results as to mortality are less than the incidence of malignancy."

FRACTURE SERVICE IN A SMALL GENERAL HOSPITAL*

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Inquiry concerning the facilities for adequate care of fracture patients in small rural hospitals has led to the conclusion that such service is not always available. While much has been said and written about fracture treatment, these contributions have been made by the leaders in this work, men of experience who are associated with the larger institutions, and lately they have stressed the importance of proper fracture therapy in small hospitals. Why are some of the major medical and surgical organizations of the nation studying the fracture problem so thoroughly and making an effort to reach the small institution with their educational campaign? Evidently these committees believe some improvement is needed. Our own conclusion, which is based on personal observation, reports from the profession, and reports of insurance companies who carry public liability and workman's compensation, is in accord with that of the fracture committee. In view of the interest in this subject, I would like to present the point of view of the small hospital.

For twenty-five years the writer has been on the staff of one of these country hospitals and for the past fourteen years has conducted a fracture service which resembles in some respects the type of service which is advocated. My object, then, is to tell the story of a small institution which has dealt with the fracture problem in its own way, to tell why and how this was done, the conditions before and after inauguration of the service, the physical equipment, methods of handling patients, and the results.

The place in question, the Emergency Hospital at Easton, Maryland, is a fair example of the average country hospital. It is small, 70-bed capacity, staffed by local physicians, situated in an agricultural community with no large industries, and admits annually a small number of fracture patients. At first thought one would not consider our fracture problem an important one because of the few cases treated. This fact for a long time dulled our sense of responsibility to these patients and it was not

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until our attention was called to some of our unsatisfactory results that we realized our shortcomings. In our hospital, as in most of the institutions of this type, the treatment of fractures for years was not given special consideration, each man treating those occurring in his practice with no one paying much attention to this work, and it was taken for granted that anyone who sustained a major fracture was destined to carry some deformity or disability for the rest of his life.

Fifteen years ago we instituted a monthly clinic for crippled children and other orthopedic patients, and, for the first year, our consultant's time was partly consumed by operations on ununited and improperly reduced fractures. For example, in 1921, five fractures of the femur in adults were admitted and four of them came to open reduction and wiring after weeks of unsuccessful treatment. Following this, Dr. Robert W. Johnson, Jr., who conducted the clinic, suggested to the staff that our handling of fractures could be improved upon and discussed with us the advisability of appointing one staff member for fracture service. The reason for this suggestion was that no one man treated enough fractures to stimulate any keen interest in the work. If one person is in charge of the department, he will be interested, can visit other clinics and keep himself informed generally as to approved methods for the handling of fracture patients. The other men agreed to refer all fracture cases to this one and allow him to accept full responsibility for their care. The necessary equipment of frames, splints, fracture table, and bed-side x-ray unit was procured, a room near the receiving ward set aside for a fracture room, and Dr. Johnson promised to stand by with advice and assistance whenever necessary. In the early days of our clinic this encouragement was a most valuable asset. Without it our service would have had a short existence.

Except for automobile accidents, which at present account for 16 per cent of our cases, there is no one factor responsible for the manner in which the fractures are sustained. They are the result of the usual accidents of civil life, falls down stairs or on the street, farm accidents, playground injuries. In the last fourteen years the clinic has admitted and treated over two thousand fracture patients. We have selected the last one thousand consecutive cases for a brief statistical review. In this series there are 679 fractures of extremities: of these, 153 were of the femur and 44 were of the inter-trochanteric type. Most of these patients were treated by either traction or spica cast and frequently a combination of the two. For the past three years about 50 per cent of the inter-trochanteric fractures had the well-leg splint applied. It has helped to prevent shortening and the patients enjoy the freedom of a wheel chair while under treatment. There were 26 fractures of the neck of the femur, the age of these patients ranging from 55 to 99 years. Except for those cases where debility and intercurrent disease made recovery unlikely, the Whitman spica was the method of treatment and has given us strong union and good position in forty-five per cent. After we learned how to secure better reduction of these fractures, and our anteroposterior and lateral x-rays showed more accurate apposition of the fragments, the results were proportionately improved. During the last year we have tried the blind method of inserting the Smith-Petersen nail and believe it will be our choice of method in the future.

The shaft of the femur was broken in 83 instances. One patient in this series had an open reduction; there were no cases of non-union; the treatment in 90 per cent of the adults was some form of skeletal traction or fixation and the results, while not always good anatomically, are much more satisfactory as to function than in the years prior to the operation of the clinic. In the treatment of this type of fracture we have watched the evolution of traction from Buck's extension to bone calipers, Kirchner wire and the Steinman pin, and in turn have used them all. Our first experience with calipers dates back to thirteen years ago, and since that time we have used some form of skeletal traction in 148 cases with nothing to make us believe the procedure especially hazardous. At present we are using skeletal fixation combined with a plaster cast. Patients treated in this manner are apparently quite comfortable, there is little likelihood of displacement, they require a minimum of nursing care as compared with the suspension and traction method, and their period of hospitalization is materially shortened, which is an important item in institutions of limited capacity.

Both bones of the lower leg were broken in 86 cases; 24 of these were treated by the double Steinman pin and cast method. No case of non-union requiring operation, no instance of appreciable deformity or disability has followed

this method of treatment. I am aware of the criticism that has been made of the steel pin method of treating fractures of the lower leg, but in the cases of severe comminution and those with long, spiral oblique fractures, the pins have given excellent results. This method is not used when fractures can be reduced and held in position with a cast, but when we must choose between a probable gross deformity or insertion of pins, our choice is the latter.

We had 106 fractures of the humerus and some form of traction, either ambulatory or with suspension from a Balkan frame, was used in 70 per cent. We had one case of non-union requiring operation, one case of amputation at the shoulder, made necessary by the destruction of the blood supply either at the time of injury or during transportation to the hospital, two patients with paralysis of the hand and wrist which could not be corrected by subsequent operation. Three patients sustained fracture dislocation requiring resection of the head of the humerus.

Fractures of the elbow in children were responsible for 63 admissions to the clinic. As far as our follow-up records show, we have two cases of appreciable deformity or disability. In one, the external condyle did not develop properly due to epiphyseal injury; the forearm is abnormally abducted but function is good. The other failure was a compound, supra-condylar fracture with subsequent infection of the joint. Motion is limited to about 25 per cent of normal.

All these elbow fractures are reduced under general anesthesia. After manipulation and reduction, we routinely take x-ray films in two positions before any splint is applied as fluoroscopic control is not satisfactory in our hands. We keep the patient anesthetized, over-expose the film, and develop quickly. Any slight displacement can be seen clearly in a film and the necessary correction made. A posterior plaster splint with a basket type of reinforcement, leaving the inner aspect of the elbow exposed, is used most frequently, and patients are required to stay in the hospital at least twenty-four hours for observation.

A small but difficult group of patients, 20 in number, are those adults who have sustained fracture of the shaft of both bones of the forearm. We have tried various types of traction splint with poor results. We have used plaster casts frequently, and open reduction, when resorted to, has not been entirely satisfactory. In our effort to find a method which would reduce and hold these fractures, we have made our own apparatus, which is cheap, easily constructed, and can be attached to any Hawley table. The patient lies on his back, the elbow flexed at a right angle, the forearm being in a vertical position, is relaxed, can be rotated, manipulated and easily viewed fluoroscopically. A half pin is inserted in the radius and a pin drilled through the ulna near the olecranon. In most cases good reductions can be secured and, by incorporating the pins in the cast, can be held until union is complete. Since the adoption of this method no open reductions have been necessary, and no failures have been noted.

No new method, no modern gadget can be substituted for good reduction. Anatomical reposition followed by proper immobilization, frequent observation and follow-up service are the points we have tried to keep in mind. To conduct such a service in a small place means hard work and long hours, but I have practiced no branch of medicine or surgery where the drudgery of detail pays greater dividends than the treatment of fractures.

Most of the fracture patients are received in a comparatively short time after injury and we are able to do early reductions. On admission, the patient, unless a condition of shock makes it unwise, is placed on the fracture table, x-rays are made and a plan of treatment decided upon. If, for example, after examination some type of skeletal traction or fixation seems advisable, this is done immediately.

All fracture patients are given a physical examination, routine Wassermann, and the facilities of the hospital are available for any other patient. The fracture chart, as approved by the American College of Surgeons, is used routinely. Serial x-ray studies are made when necessary and no fracture is reduced without x-ray examination. After reduction, all fractures are x-rayed in two positions or stereoscopically.

The responsibility for proper treatment of fractures in the South rests heavily on the Bone and Joint Section of the Southern Medical Association. More can be accomplished by your individual guidance and assistance than by fracture committees and hospital surveys, although I do not minimize the importance of these organizations when and if they function. You are recognized as authorities on this subject and your opinions are respected by the rural hospital staff and the physicians who refer patients to them. Not only will your advice be valuable

to any clinic but your endorsement will insure for it the recognition necessary for successful operation. In the small hospitals which have met the minimum requirements of the American College of Surgeons, fracture treatment has only recently been given serious consideration. The rules of such hospitals require that most surgery be performed only by those who have been properly trained, but there has been nothing to prevent anyone from treating severe fractures. At present, the fracture committee, which includes some members of this section, is attempting to develop some suggestions and regulations for fracture treatment and give this work a measure of the importance it deserves.

Concerning the transportation of the fracture patient to the hospital, we have made little or no progress. The ambulance drivers and the fire engine crews have received instruction in the use of traction splints, but in 90 per cent of automobile accidents none of these facilities are at hand. The patient is hurriedly placed in the first automobile to reach the scene and rushed to the nearest hospital. Frequently he arrives in a condition of shock exaggerated by a ride over rough roads at high speed. We know of no way to control this. The only suggestion is educational propaganda among the school children, with lectures, and slides illustrating first aid, and this we expect to accomplish in the near future. The children will be taught how to utilize whatever is available for splinting. While the use of the Thomas splint is ideal, some of the best examples of emergency splinting seen by us were furnished by Boy Scouts and telephone crews who knew how to use palings from fences, saplings or pieces of board. In a rural section we believe this sort of first aid instruction will be more valuable than well trained ambulance crews that are not on the spot.

A subject which is of vital importance is the cooperation of the hospital staff in referring fracture patients to the clinic. In our hospital we have been fortunate in that most of our colleagues demonstrated a high type of cooperative spirit and willingly relinquished their patients to the fracture department.

SUMMARY

As two-thirds of all major automobile accidents occur in rural districts, it is necessary that the small hospital treat an increasing number of fracture patients.

Better care of these patients can be provided by a definite fracture service or clinic.

The necessary equipment need not be elaborate or expensive and is well within the financial limits of most small institutions.

An orthopedic consultant can help materially in building up these clinics because of his acknowledged position of leadership in fracture work.

The principal reason for this presentation is to suggest that you, as orthopedic surgeons, use your influence, offer encouragement to the small hospitals and aid them in establishing a fracture service wherever interest is shown.

I believe an investigation of the fracture clinic as operated for fourteen years at Easton will demonstrate the possibility of improving fracture treatment in the small country hospital.

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DISCUSSION (*Abstract*)

Dr. H. Earle Conwell, Birmingham, Ala.—It behooves every man doing fracture work to see that his fracture service is well organized, that all material is centralized and that one individual in the hospital, preferably a very capable orderly, be responsible for all fracture equipment. Nothing is more pathetic than improperly applied traction apparatus or any other fracture apparatus.

No surgeon responsible for fractures can wait until the fracture arrives at the hospital before looking for the equipment to treat it. This is as absurd as for the surgeon to attempt abdominal work without proper instruments available before the patient arrives at the hospital.

Dr. Hammond has spoken mainly of the small hospital. What he has said as to the requirements of fracture service in a small hospital is also true and necessary in the bigger hospital, but they are often lacking.

Too much first aid treatment by the layman is not desirable. However, we will have to admit that there are certain lay members who in many instances are as capable as many physicians in carrying out first aid. The future of first aid looks bright so far as teaching the layman first aid methods, especially when we realize that each year thousands of Boy and Girl Scouts are being trained properly along such lines. I believe that within another generation, and it will take as long as that, the public will be more conscious of first aid in fractures as the result of this move with the Boy and Girl Scouts.

Dr. Robert W. Johnson, Jr., Baltimore, Md.—I am familiar with the fracture clinic which Dr. Hammond has built up at the Easton Emergency Hospital, and consider that the treatment and results there are as good or better than those to be found in large medical centers. He has conclusively demonstrated how the work in smaller communities can be done. Several factors have been essential to the success of his clinic. The first is a hospital staff matter; the rest are personal qualities.

(1) Concentration of all fractures into the hands of one man who assumes their responsibility and the bur-

den of the work, but in return gets volume enough to give him experience in the care of fractures. This requires the active cooperation of the other physicians in the community.

(2) Enthusiasm. The realization that the proper treatment is a difficult job, testing the skill and ingenuity of the doctor and making it a game of wits, the winning of which is a great satisfaction aside from any monetary reward, which in the case of fractures is usually very meager. It must almost be regarded as a professional hobby.

(3) Mechanical skill to accomplish results rather than the traditional surgical or operative point of view. As a matter of fact, it is of interest to realize that Dr. Hammond was an internist and roentgenologist and only became involved in this fracture work through x-ray.

(4) Patience, not only in the work with individual patients, which is a practical necessity, but in slowly demonstrating to the community at large that hospitalization and adequate care are fruitful in reducing to a minimum the bad results they have become used to accepting as inevitable in almost all bad fractures.

I believe Dr. Hammond has presented here to us a carefully worked out and proven experiment in the care and treatment of fractures, which should have wide application and be productive of an inestimable benefit to both doctors and patients in rural or small communities.

ACUTE INFECTIONS OF THE MIDDLE EAR AND THE MASTOID CELLS*

By JOHN J. SHEA, M.D.
Memphis, Tennessee

Acute infections of the middle ear and the mastoid cells have as their complications the most serious conditions that arise in the practice of our specialty. Unfortunately, these complications frequently develop in our most important cases. Fraser and Halliday[1] reported 139 (15 per cent) intracranial complications out of 891 consecutive cases of acute mastoiditis from the Royal Infirmary of Edinburgh during the past fifteen years, of which sixty (6 per cent) ended fatally. Boies[2] reported 300 cases of acute mastoiditis from his private practice and the otolaryngological service of the University of Minnesota Hospital of which the incidence of extradural inflammation was 35 per cent. The distribution as to the site of the extradural pus was as follows:

Location	Number	Per Cent
The lateral sinus	64	21.6
The middle fossa	17	5.6
Both lateral sinus and middle fossa	17	5.6
The surface of the petrous pyramid	7	2.3

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An abscess of the ear should never be considered an uneventful but unfortunate event in the life of a child, as it may be the beginning of such important complications as thrombophlebitis of the lateral sinus, meningitis, brain abscess or petrositis. The three most common pathways of such an intracranial extension are through the tegmen of the middle ear or of the mastoid antrum and through the bony wall over the lateral sinus. The more rare routes are along the nerves at their exitus through the internal auditory meatus or extension through the cells of the petrous process. An infection of the middle ear readily passes through a fissure left after a fracture of the base of the skull, if the fracture extended into the middle ear or the mastoid cells. On the other hand, the ears cannot be blamed for all intracranial infections, for in some of them the infection has extended from the sinuses, notably the sphenoid. Dwyer[3] contends that

"We must change our conception of these cases. I had always thought that the infection traveled in from the mastoid. I have come to the conclusion that a large majority of our mastoid deaths are really not due to the mastoid. In other words, there is an extension from the sphenoid process, probably in the form of a low grade osteomyelitis or a low grade thrombophlebitis, which causes the meningitis, rather than extension from the mastoid or mastoid surgery itself. Two cases in which I did not have the opportunity of doing a complete autopsy, but got the bones out after death, proved that point to my satisfaction."

There has been a tendency in the past to separate the acute infections of the middle ear and the mastoid, but today we appreciate their continuity and the designation of an acute middle ear or an acute mastoiditis are but different chapters in the cycle of the same inflammatory process. The mastoid cells bear a constant relationship to the middle ear as do the paranasal sinuses to the nose. At the onset of an acute infection within the middle ear there occurs a protective reaction within the mastoid designed to aid in the immunization against the invading organism. Nature intends the mastoid cells for some purpose and an appealing elucidation of their existence is to assist in the combating of infections. The most logical explanation why some infections almost reach the point of mastoid cell coalescence, to have a sudden change for the better, and end in recovery, is that the local process of immunization improved sufficiently to wipe out the advancing disease.

It is not intended in the scope of this paper to deal in an academic manner with the etiology, pathology and symptomatology of acute infections of the middle ear and the mastoid cells,

but rather to present clinical impressions that have served the author as a working knowledge in the treatment of infections of the middle ear and the mastoid cells.

MEMBRANA TYMPANI

The first aural manifestation in influenza may be blisters upon the posterior superior part of the membrane, which at first contains a serous fluid, but which rapidly becomes hemorrhagic. This condition must not be confounded with a herpes oticus, for this latter condition may be associated with severe vertigo and permanent loss of hearing. The formation of these blebs is accompanied by severe pain, which is relieved by their destruction. Care should be exercised that the drum be not perforated, for the disease is superficial and the middle ear at this time may be free of infection. The blisters may be destroyed by rubbing them with a cotton covered applicator soaked in a solution of cocaine. If left alone the pain continues until spontaneous rupture occurs, but occasionally a perforation will result with a contamination of the middle ear. These blebs should not be confused with the exudation sacks seen at later stage in adults with a true middle ear abscess. This latter condition develops in the posterior superior quadrant when the drum has been thinned by previous middle ear catarrh (Politzer[4]).

OTALGIA

The brain is protected from insult and injury by a thick skull and the ear by its sensitive nature. The pain of an acute infection of the middle ear may be piercing and radiate towards the vertex, neck and teeth and be accompanied by a violent headache. Pain is more severe in a child than in an adult, but is seldom continuous, being worse in the evenings and during the night. On the other hand, the remission of the pain in the child is more common than in the adult. The internal administration of one of the derivatives of opium for the relief of the pain is preferable to the sedative effects of the barbiturates. The pain of intracranial extension is more severe than that caused by inflammation of the bone. The experienced nurse will report that the cry of the child has changed when the infection extends inwardly involving the meninges.

Ruttin[5] taught that:

"The patient with meningitis accented the last syllable in his yell, while in brain abscess the patient accents the first syllable."

MYRINGOTOMY

It is best to avoid early myringotomies, in order to allow the middle ear to develop protective barriers against the infection. On the other hand, early incision of the drum is the practice of some authorities as an abortive measure and when successful the disease is of short duration, but when it fails, it delays the reparative process. If a myringotomy is performed, the use of sodium citrate wicks will prevent the coagulation of blood or serum over the wound, thus reducing the necessity of repeated incisions. A strong band of radial fibers exists in the posterior light reflex, though having nothing to do with it. These fibers have an antagonistic action to the tensor tympani muscle. An incision through these fibers makes a more permanent opening due to the contraction of the fibers and there is less chance of injury to the chorda tympani nerve in this locality than when the incision is made higher along the posterior canal. The necessity for repeated myringotomies is an indication of a surgical mastoid.

MASTOIDITIS

When does the otitis media become an acute mastoiditis? If the initial pain was relieved by the establishment of aural discharge, but returns after an absence of several days, it is an indication of an extension of the disease from the middle ear into the mastoid cells. The same may be noted regarding the temperature, though an excessively high temperature is not a requisite for the diagnosis of a surgical mastoid. The physical examination usually reveals some diagnostic feature, the progressive sagging of the posterior superior canal wall is suggestive and some swelling over the mastoid process occurred in half of my youthful cases. The absence of tenderness over the mastoid antrum or at the tip is not negative proof against the existence of an acute mastoiditis. The continuation of profuse purulent discharge indicates that a surface of sufficient area must be involved to produce such a quantity and certainly the middle ear is too small to be blamed. Massive evidence such as external rupture, acute facial paralysis or severe dizziness occurring during the course of an acute middle ear infection clearly establishes the presence of an acute surgical mastoiditis.

The hematological study should present an increase of the total amount of the leukocytes with a shifting to the left of the Schilling differential count during the progressive stage, with a steadying, then a shift to the right as the immunity is established. A total white count of over 20,000 in an acute mastoiditis demands the exclusion from the diagnosis of pneumonia and brain ab-

scess; for severe cases of meningitis have a count only around 10,000. The red blood count is that of a secondary anemia, especially when the infecting organism is of the hemolytic type of streptococcus. The bacteriological study includes most of the membranes of the pyogenic group, especially the streptococci. The streptococcus mucosa is the most dangerous and when present the surgeon should anticipate complications.

Repeated roentgenograms of the mastoid are valuable in following the extension of the disease within the cells, but the clinical picture must be constantly compared with this study when crystallizing the opinion.

When to operate cannot be determined by a cold rule of thumb. It is better to wait a day or two longer than to operate prematurely, but when the surgeon considers his patient as not doing well after thorough conservative treatment, then operative interference is indicated. The demand for early surgery is more frequent in the cases where the infection has had a sudden onset with violent symptoms and a rapid involvement of the mastoid process.

Case 1.—J. M., aged 7, was seen in consultation forty-eight hours after the onset of an acute scarlet fever and operated upon that day. The pediatrician gave the history, that the child became ill the morning of February 15 and the family summoned him at noon. His first examination revealed that the lad had an acute febrile disease with a very red throat. During the first evening of his illness, the child complained of a sudden severe pain in the left ear, which demanded the physician's return. Examination showed an acutely inflamed drum, which upon incision allowed the escape of a thin purulent discharge. A rash developed the second day and a diagnosis of scarlet fever was made. The ear continued to pain the lad in spite of the free drainage, and the discharge became purulent. I saw him the following morning, found that he had the signs and symptoms of a fully developed acute mastoiditis and advised his transfer to the hospital. That afternoon a simple mastoid operation was performed and the gross findings were:

"The canal was filled with a purulent discharge; the soft tissues over the mastoid process were edematous; a small perforation was visible over the mastoid antrum; the coalescence of the mastoid cells was visible over the mastoid antrum; the coalescence of the mastoid cells was greater than expected and there was an exposure over the lateral sinus."

The postoperative convalescence was assisted by a transfusion and the lad made an uneventful recovery.

Here is an instance where, forty-eight hours after the acute onset of a middle ear infection, there was an external perforation and a loss of the inner table. How long would it have required for this lad to have developed an intracranial lesion at the rate of progress of the disease? Probably only a day or two more.

SUPPORTIVE MEASURES

The virulence of the organism and the resistance of the individual with a normally pneumatized mastoid process are the two most important factors in determining whether the mastoiditis will subside or require surgical interference. We have no means of changing the virulence of the organism, but there are many supportive measures at our disposal. Daily injections of modified bacterial antigens with bile lipoids and neutral fats during the acute infection of the middle ear have served me best. Vaccines are too slow in their action for such an acute affair. Streptococcus antigen, administered prior to the operation, fortifies the tissues against the overwhelming invasion as the result of surgical trauma. Blood transfusions are popular in their preoperative use as an immediate tonic. A well-timed transfusion increases the immunity of a child with an acute mastoiditis and may assist in its recovery without surgery. Likewise, a transfusion is equally valuable for a preoperative patient in a weakened condition. If the hemoglobin has been reduced to 75 per cent the demand is imperative, but irrespective of the white blood count with its differential or the red blood count with its hemoglobin content, a transfusion should be administered before or after the operation, when the patient's condition is not satisfactory to the surgeon. This procedure reduces the frequency of complications.

INTRACRANIAL COMPLICATIONS

Meningitis and sinus thrombosis are the two fatal intracranial complications of acute infections of the middle ear and the mastoid cells. Meningitis may be limited to the middle fossa and present only the initial symptoms of headache and fever, while meningitis of the posterior fossa in 95 per cent of its instances is an extension of a labyrinthitis and is accompanied by vertigo with nystagmus. Unfortunately, meningitis develops during the early stage of some infections and its presence prior to operation defeats the purpose of the surgery. Such an instance was as follows:

Case 2.—B. L. G., aged 6, was admitted to the hospital on January 2, 1936, with an acute left mastoid, in which pain was the most prominent symptom and was characterized by its radiation from the ear to the occiput. The ear was examined and the canal was seen to be filled with a purulent discharge. There was a perforation in the posterior half of the drum, with a sag-

ging of the superior canal wall. The roentgenologic report showed the mastoids well developed; right cells clear; left cells cloudy; cell delineation obscured with some very probable erosion of cell structure. The diagnosis was left mastoiditis.

The blood count showed 3,500,000 red cells, hemoglobin 69.6 per cent, white cells 15,600, polynuclear 86 per cent, and lymphocytes 14 per cent.

During the first day of hospitalization the temperature ranged from 104° on admission to 101° with a corresponding pulse rate. On the fifth hospital day, an opinion was given that the mastoid was surgical. A consultation was held, in which a request for further local treatment was demanded. On the tenth hospital day, surgical interference was unanimously decided upon, but the child's condition on the morning set for the operation was such that it was decided to give him a transfusion before the operation. By midday a left-sided facial paralysis developed. At operation that evening the mastoid was found to be normally pneumatized, a subperiosteal abscess was exposed, pus and granulations filled the cells. The anterior and posterior group of cells were followed deeply. The child reacted normally after the operation, but the next day presented a rotary nystagmus and complained that pain extended down the left side of his neck. Seventy-two hours postoperatively his temperature had risen to 101°, the child became very fretful and a neurological consultation confirmed the diagnosis of beginning meningitis. The lad developed a fatal meningitis, the offending organism being *Streptococcus hemolyticus*. Five transfusions averaging 300 c. c. of whole blood were given during the course of his meningitis. Continuous spinal drainage was maintained after repeated spinal tappings had failed to control his meningeal symptoms.

This child apparently was doomed to meningitis from the first examination made on his entrance into the hospital and he had the typical cry of a meningitis victim throughout the last three weeks of his illness.

THROMBOPHLEBITIS OF THE LATERAL SINUS SECONDARY TO MASTOIDITIS

Sinus thrombosis is formed by an extension of the inflammation from the mastoid cells into the lateral sinus. In the hemorrhagic type of mastoiditis the extension is through the blood vessels and the clot is continuous from the thrombosed vessels of the bone into the sinus proper. The inflammation may extend directly by a necrosis of the bony wall of the sinus in the coalescent type. This latter method produces two varieties of perisinus abscesses. The first is composed of a collection of pus on the outside of the sinus wall, having no connection with the mastoid cells; while in the second variety, the pus is connected with that in the mastoid cells. The thrombus within the sinus may be limited to the sinus wall and not obstruct the lumen of the vessel, or it may be completely obstructing the lumen and extend backward to the torcular or downward through the bulb into the jugular vein and even into the heart. Septicemia is the outcome of such a complication and the future course of the disease will be septic.

Unfortunately, these patients express a feeling of well-being during the onset of the formation of the thrombus. As with phlebitis elsewhere, many recover spontaneously, but untreated the majority will develop meningitis or produce fatal metastases in the lung, liver, kidney, muscles or joints. Occasionally, the formation of a metastatic abscess serves as a filter and clarifies the blood stream. The differential diagnosis of this complication is assisted by a study of the optic disc, for sinus thrombosis will choke the disc. The spinal fluid is clear in thrombosis and cloudy in meningitis. The number of cells remains within the normal limits in contrast to the great number in meningitis. Likewise, a culture of the spinal fluid should fail to show any growth of pathogenic organisms. The treatment demands an exposure of the sinus. If the sinus is normal in appearance one may procrastinate before opening it, but if it is abnormal, the sinus should be aspirated or incised to ascertain the presence and type of the clot. The further surgical management may require a ligation of the jugular vein with emptying of the clot and the packing off of the sinus.

BRAIN ABSCESS

Extension of infection from the temporal bone is the cause of 83.2 per cent of acute brain abscesses (Evans[6]), but the warning of Dwyer as given is to be remembered: "The importance of the sphenoid sinus as a causative factor must be considered."

Acute brain abscess complicating acute infection of the middle ear and the mastoid cells presents a neurological problem greater than the management of an acute or chronic brain abscess accompanying a chronic infection. An acute meningitis or thrombophlebitis is more likely to develop as the result of the extension of an acute process than the formation of an acute abscess. When an acute abscess does develop, it has no definite wall and consists of a broken down area of brain tissue with a central area containing pus and detritus. An edematous process surrounds the inflammatory area and not until the abscess becomes chronic is the so-called capsule formed. In many of these cases, there is an extradural collection of pus over the location of the acute brain abscess.

The controversy as to the best method of han-

dling brain abscesses is beyond the scope of this paper.

CONCLUSION

The complications of the middle ear and the mastoid cells are the most frequent causes of death met with in the practice of otolaryngology and their frequency is greater than is appreciated.

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DISCUSSION (*Abstract*)

Dr. Edward A. Looper, Baltimore, Md.—One of the most satisfactory accomplishments of modern preventive medicine has been in the practical reduction of ear complications.

A great deal of this credit must be given to the routine examination of the ears, nose and throat of school children. Thousands of children have been saved the seriousness of otitis media and possible mastoid involvement by early removal of tonsils and adenoids, as a result of such investigation, and parents have benefited by this plan of education, which would never have been accomplished in any other way.

Enough time has now passed to demonstrate the results of this work, for everyone must admit that acute ear infections and mastoid involvement have become decidedly less in the past few years. Another factor of importance is better recognition by the profession in general of early ear symptoms with an immediate paracentesis. Few physicians are now indifferent to the symptoms in a child who complains of a painful ear, deafness, temperature and a red ear drum. They do not wait for a drum "to break" with an irregular opening which may never heal, but appreciate the importance of a clean crescentic incision which closes without complications. With modern electric otoscopes the ear drum can be clearly seen under all conditions and the operation can be easily performed with the proper training. But, on the other hand, a careless paracentesis may do irreparable damage to the ear drum which may affect the hearing throughout life. So this operation should always be performed by one who is skilled in its technic.

In most cases early paracentesis prevents serious mastoid complications and should be performed in all cases of acute suppurative otitis media. In a few patients mastoid infection will develop regardless of all precautions. Such cases demand a knowledge of the pathological changes occurring in the mastoid antrum and cells. X-ray is of little value in the first week or ten days, but is of great importance after the second week. Most cases of simple otitis media will clear up in two weeks, but if symptoms persist after this time, mastoid involvement is to be suspected, and immediate drainage is indicated. Continued temperature, pain and high leukocytosis after a paracentesis should lead one to suspect extension of the infection. It is not necessary to wait for edema over the mastoid before suspecting infection in these cavities, because edema usually means that suppuration has broken through the periosteum externally, fortunately, in most instances, because if by chance the infection passes through the internal table, meningitis with death will result.

In my opinion, every patient with a discharging ear lasting more than two or three weeks should be considered a potential mastoid complication, and unless proven otherwise, should at least have an inspection of the mastoid area. To me the seriousness and severity of this operation has been over-emphasized, for a simple mastoid operation is really simple and if more operations were performed when so indicated, I am convinced that we should have many fewer chronic discharging ears with useful hearing than we have today. Purulent infection indicates drainage, so why not play safe and give the patient the benefit of the doubt instead of procrastinating with a condition which may mean the patient's life, or at least mean impaired hearing with later a possible radical operation.

The delayed acute cases are those in which sinus thrombosis is most apt to occur. Even in this most serious involvement definite clinical signs indicate what is going on. A chill is always a dangerous symptom in ear cases. It means pyogenic infection close to or in vital structures; most often it means invasion of the blood stream by infected material and unless the focus is removed, prognosis is grave. Prompt attention with ligation of the venous supply will be the means of saving the patient's life in most instances. Therefore, in conclusion: Brilliant results have been accomplished in otology by better education of the public as to the predisposing causes and proper elimination of factors producing ear infections.

Physicians should always be on the alert to recognize early ear symptoms and should recognize their responsibility in seeing that their patients have the best advice available of a competent otologist who is capable of dealing with all complications.

MICROSCOPIC ANATOMY OF THE TEMPORAL BONE SHOWING PATHOLOGIC ROUTES OF EXTENSION TO THE CRANIAL CAVITY*

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The grave consequences of disease in the temporal bone are conditioned by the anatomy of this region. Whether an existing pathologic state is primary or secondary or is concomitant with systemic diseases such as essential hyper-

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tension, diabetes, syphilis or tuberculosis, this fact remains. The close relation, both anatomically and phylogenetically, of the tympanum to the nasopharynx with its susceptibility to infection augments the difficulties. The poorly ventilated cavities of the tympanum, attic and mastoid with their moist membranous lining afford excellent culture media for certain types of bacteria. The temporal bone, forming, as it does, an extensive part of the floor of the cranial cavity and lying in intimate relation with important venous channels, holds a strategic position in the battle for the maintenance of the health of the individual.

Infection within the body may spread by (a) vascular metastasis, (b) direct extension, (c) a combination of these two processes. As elsewhere in the body the early appearance of a pathologic picture is intimately associated with the vascular supply. Three major conditions present themselves as prerequisites to a diseased condition: (1) Blood may fail to come to the region because of trauma, emboli or thrombi with the result that an anemic infarct may develop. This condition occurs where vessels are terminal with little or no anastomosis of the arterial capillaries of one artery with those of another. An example of this may be seen in the inner ear. This organ is supplied solely by the internal auditory artery, a branch of the basilar which, as we know, is formed by the union of the two vertebral arteries. No collateral circulation exists to take care of the situation in cases of obliterative endarteritis, for example, or thrombi. (2) Blood coming to the region may be deficient in one or all of the following: oxygen, antibodies and nourishment, or it may come laden with bacteria. (3) Metabolic waste products may not have been carried off so that these accumulate to destructive proportions.

Vascular changes such as obliterative endarteritis, atheromas, arteriosclerotic plaques, emboli, thrombi and venous stasis occur within the temporal bone region as in any other organ of the body. For this reason it is important to review the anatomic vascular anatomy.

As S. Ruskin has said, the temporal bone lies in a venous bed. This consists of the sigmoid sinus, jugular bulb, superior and inferior petrosals, pericarotid plexus and cavernous sinuses. The symmetrical schema is true, however, for only 3 per cent of the cases. In 70 per cent of the cases the right lateral sinus receives the blood from the superior longitudinal sinus, while the left lateral drains the smaller but equally important straight sinus. In addition to these wide, tortuous and somewhat sluggish venous pathways, numerous minute veins must be considered. Streeter has shown that in the human embryo the saccus endolymphatics is enmeshed in veins many of which empty into the lateral sinus. In the second month the saccus itself is closely applied to the wall of the lateral sinus. This juxtaposition is lost with development, but the venous network persists. Anson and Nesselrod have recently described the vascular supply of the adult saccus as similar to that of the intestinal villi and state that it is less abundant in the terminal of the three sulci of the saccus.

Other veins of importance, although small, are the numerous venae comites accompanying the arterial vessels of the temporal bone. These drain into the various sinuses listed above. The veins of the subarcuate fossa come under this category.

The arterial supply of the middle ear and mastoid comes from four distinct and sizeable arteries. These are (1) the internal maxillary via the deep auricular, anterior tympanic and middle meningeal; (2) the posterior auricular via the stylomastoid artery; (3) the ascending pharyngeal via the inferior tympanic artery, and (4) the internal carotid. Within the tympanum, mastoid and tube the terminal branches of these vessels undergo rich anastomoses.

A branch of the middle meningeal artery anastomoses with the stylomastoid artery within the facial canal. The venae comites of these two vessels do likewise. The anatomic fact that the bony facial canal is not a closed canal, but is, constantly in the infant and child, and occasionally in the adult, open to direct extension of pus from the niche of the oval window, must be recalled. This aperture in the facial canal, as has been previously mentioned by the author, is of phylogenetic interest since it is a hangover of the period when the stapedial artery was an important functional aortic arch.

The blood supply of the bony capsule of the human fetus has been studied and modeled by Bast. In the capsule we apparently have very little anastomosis of terminal branches, a fact of probable significance, particularly where the region of the fissura antefenestram is considered. The abundance of hematopoietic marrow in the petrous apex of the infant and child and frequently in the adult must be borne in mind. There is a veritable bed of marrow surrounding

the cochlea in young infants and this is directly continuous with that of the petrous apex. In many places this apical marrow is exposed to direct communication with the middle ear mucosa. In cases of infection an osteomyelitis may develop here. This may persist after the primary focus of infection in middle ear or mastoid has cleared. Vessels taking origin in this region would, of course, disseminate infection.

Following the excessive accumulation of waste products, bacteria and pus, nature employs various methods to rid the region of disease. An attempt may be made to wall off the offending area, frequently with worse consequences at a later date. During this process the vascular system is again called into activity as granulation tissue develops. Through numerous newly formed vessels the disease may be spread by metastasis to the brain or to more remote organs, the kidney, for example.

In the event that nature fails to wall off the disease or rid the temporal bone of the offensive condition by metastasis, she next resorts to direct extension.

Eagleton, in his book on cavernous sinus thrombophlebitis, states that "by far the larger proportion of all cases of brain abscess and meningitis are caused by the infective process traveling by way of an infective retrograde thrombophlebitis and not by direct extension of the suppuration." Microscopic sections of the temporal bone, however, certainly show evidence of both routes. Eagleton himself states in his discussion on perivasculitis that "the outer fibrinous coat of the sinus is first attacked by microorganisms from an adjacent bony caries or a perisinus abscess." Further, that "parietal mural clots arising from adjacent bony caries are very common in the lateral sinus." Obviously the thrombotic condition in such cases arises from direct extension.

Routes of direct extension may occur from the middle ear cavity into the inner ear, thence to the cranial cavity or directly from the middle ear cavity with or without involvement of the inner ear. The routes from the middle ear cavity to the inner ear have previously been reviewed and illustrated in a paper by Dr. Dean and myself. Briefly, they include fistulae of oval and round window, canals or cochlear wall.

Once the infection has reached the inner ear, it may proceed to the cranial cavity by various paths. The cochlear aqueduct leads off to a cisterna from a depression just one millimeter within the round window membrane. This cisterna is in direct communication with the subarachnoid spaces. If the oval window is the point of entrance the endolymphatic ducts lead off from the vestibule just opposite the footplate of stapes. The cribriform areas through which the branches of the eighth nerve enter probably form possible paths of extension and the author has occasionally seen at autopsy a mass of pus just at the internal auditory meatus. The three routes mentioned might one or all be routes for vascular extension as well as direct.

Routes without necessarily involving the inner ear will now be considered. Carious dehiscences may occur in the tegmen of middle ear or mastoid. Fistulae may occur in the hypotympanum. In young infants the dura covering sutures and fissures which are closed in later life may give way and allow the direct extension of pus. Hyrtle's fissure is one such channel. This fissure runs from the niche of the round window infero-medially in a direction practically parallel with the course of the cochlear aqueduct. Another infantile route of extension is through the petrosquamosal suture directly into the cranial cavity.

As Dr. Alden's case of a 5-year-old child illustrates, fistulae may enter the apex from an infected tube. In both young and old a petrositis, or more particularly an apicitis, may lead to involvement of the dura covering the petrous tip. This may be the result of an osteomyelitis of the marrow in the region or of an osteitis of the bone covering a diseased pneumatic tip. Dehiscences may occur at the apex also with extension of pus into the region of the Gasserian ganglion. Dr. Cone's post-scarlet case is an example of this. According to Ruskin, congestion of the inferior petrosal is sufficient to give the clinical syndrome of Gradenigo.

It must be borne in mind that in addition to the six vascular routes here demonstrated, similar conditions could occur wherever there are vessels in the temporal bone. A dozen routes of direct extension have been demonstrated. Realizing that all these routes may appear in various combinations with each other, we are indeed confronted with the hazards to life in temporal bone involvement.

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DISCUSSION (*Abstract*)

Dr. S. J. Crowe, Baltimore, Md.—I wish to emphasize the danger of an infection in the petrous part of the temporal bone. It is not a rare condition and is, I believe, often the unrecognized cause of a fatal meningitis in young children and infants. Infections of the petrous pyramid occur also in adults but the diagnosis is not so difficult in adults as it is in young children. It is often impossible to make satisfactory x-rays of the base of the skull in a child without the use of general anesthesia. To interpret films of this region it is necessary to see a large number of x-rays of normal as well as of infected petrous pyramids. In addition, the subjective complaints of the adult in regard to localization of pain, diplopia, and so on, are of great aid in establishing a diagnosis.

The family physician, the medical consultant, the pediatrician and the otologist should all review the anatomy of the temporal bone and the surrounding structures and familiarize themselves with the literature of petrous pyramid infection if they hope to recognize the symptoms of this disease during its early stages, at a time when the proper surgical measures may save the life of the patient. The signs and symptoms of suppuration of the petrous pyramid and the methods used in making a clinical diagnosis were admirably summarized in a symposium before the American Otological Society in May, 1935 and are published in the Transactions of the Society and in the Annals of Otology, Rhinology and Laryngology for December, 1935.

More than half of the surface of the petrous part of the temporal bone is covered with dura mater. The petrous pyramid infections are either primarily in the bone marrow (osteomyelitis) or in the pneumatized cells. The latter are in every respect similar to an infection in mastoid cells and, like mastoiditis, are secondary to otitis media. Both types of infection may give rise to extradural abscess, thrombosis of the petrosal and cavernous sinuses, brain abscess or meningitis.

Dr. Ella M. A. Enlows, Washington, D. C.—Dr. Wolff is to be congratulated upon her presentation. From many years at the microscope I know what an enormous expenditure of energy excellent work of this type requires.

Her paper enables us to visualize with great clarity these pathways of intracranial infection and we should be more than ever willing to accept as fact the statement so often made, but too infrequently considered, that every patient with middle ear disease is seriously ill. And the facts as presented emphasize the importance of early recognition of this condition. Also, since otitis media and mastoiditis are frequently seen in the young, pediatricians should be urged to allow no unnecessary delay in calling for advice from the otologist as to alarming signs and symptoms. Early myringotomy and free drainage may prevent the increased pressure which would break down the flimsy barriers blocking entrance to the cranial cavity by the routes Dr. Wolff has outlined. Infected paranasal sinuses or throat, improperly treated or entirely neglected, may continue to send bacteria or toxic products to this already overburdened region.

It is indeed interesting to speculate upon the marvelous recovery of most of our patients in spite of the apparent ease of overflow from the middle ear and mastoid.

CONCERNING NORMS, NEBULAE AND FOREIGN BODIES IN REQUIREMENTS FOR CLINICAL CERTIFICATIONS*

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In Baltimore, this mid-November, nineteen thirty-six, are occurring two noteworthy, periodic medical gatherings. One is large, mature, and so well-renowned that every man knows and esteems it. The other is tiny; and so new and unheralded as to be known only to its interested few. The former is the thirtieth annual meeting of the Southern Medical Association, of which we here form a part; the latter is the first meeting of the American Board of Pathology, for the examination of physicians who aspire to that Board's certificate of qualification to practice pathology as a clinical specialty; and its first examination is designedly occurring at the same time and place with the thirtieth annual meeting of the former.

It were superfluous to emphasize the value of the former gathering; but it is meet to dwell, briefly at least, upon the high significance, to the lay and medical world, of the latter gathering.

The American Board of Pathology is the newest, and next-to-last to function, of a complete group of coordinate boards of an official character conceived and created by the organized physicians of America to cover the gamut of the presently recognized major fields of clinical service by affording to physicians appropriately qualified, respectively, to practice them, due opportunity to become authoritatively so certified to the public and profession.

Despite the disparity in size, age and prominence between the cited medical gatherings, there is no disparity between them in potentiality for the advancement of American medical service. This is true because of the prime importance of the integrated whole of which the

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American Board of Pathology is a self-contained part.

Specifically, there now function (in order of establishment) the American Boards of: Ophthalmology, Otolaryngology, Obstetrics and Gynecology, Dermatology and Syphilology, Pediatrics, Psychiatry and Neurology, Radiology, Orthopedic Surgery, Urology, Internal Medicine, and Pathology; and the functioning of an American Board of Surgery is imminent.

The initial functioning of the American Board of Pathology has especial modern significance as formal, national recognition of the pathologist as a major partner in the great whole of American expert clinical service.

Since the American Board of Surgery may be expected to have become fully organized in 1937, the year 1938 may be taken to mark the completion of the first stage in the evolution of a new and definite epoch in American medicine.

This first stage will have required a quarter-century for its completion. It began in 1913, when the ranking national associations of ophthalmologists united in furthering a special educational movement within their own field. It cannot be thought that the ophthalmologists in 1913 had any prescience of what was to evolve; but from their beginning did ensue a gradual evolution which is now culminating with the terminal event just stated.

However, we are nearing only the completion of a first stage in the entire evolutionary process, considered in a national sense. If we take 1938 as marking the completion of this first stage, we may also take it as marking the beginning, also in a national sense, of a second stage, logically necessary, which doubtless will require another decade for completion, let us say by 1948.

The general nature of the second stage in the evolutionary process is obvious. If in a national sense we exert powerful and righteous moral force toward certifications as to clinical expertness, we must also nationally provide educational machinery fitted to qualify younger physicians for such certification. A consideration of certain phases of this second stage is the guiding purpose of this article.

One should keep clearly in mind that this whole matter really centers upon a distinction between the voluntarily casual and the authoritatively systematic manners of doing things; or perhaps one might say, between intangibles and tangibles, or between virtualities and realities.

Casually, or intangibly, or virtually, as you will, we always have had certifications as to clinical expertness; and, in the same sense, we always have had the correlative educational machinery. Clinicians' status in their communities, their hospitals, their faculties, their societies, and with their fellows, if generally commendable, constituted their virtual certifications; and the various and indefinite instrumentalities which they severally utilized in attaining to such status constituted the related casual educational machinery.

But the history of advance in clinical service is replete with examples of progress effected by the discarding of the casual for the systematic; and that principle very properly underlies the activities of American boards for the certification of clinical specialists. Their continued activities, and their refining and seasoning of the same, forecast a status of possession of actual certification by every duly worthy and matured clinician, with the reasonably connoted assurance that he is qualified for expert service within his field of certification. Such status readily includes the case of the "general practitioner," whose essential certification is for internal medicine.

Thus, in medical progress, we have entered upon a new epoch in the conditions and qualifications for clinical service. But the picture of the instrumentalities involved, while clear and admirable in outlines, and also in some details, is void, cloudy or faulty in other details. It must be a prime business of the second stage of this evolution to refine the picture to that masterpiece quality which we may well believe to be its destiny.

There is neither purpose nor occasion herein to recite all of the facts concerning the general "Advisory Board for Medical Specialties" or concerning the several special, certifying "American Boards" (as they are currently and briefly termed). These facts are matters of readily available record. Rather it is our purpose to discuss only certain features and implications of immediate importance. In the discussion constructive criticism must appear; but it is to be understood that any shortcomings noted should be attributed to the newness of the whole movement, and that rectification as the movement matures is the justifiable prediction. Experience will disclose and provide for much that has been overlooked and unforeseen; and all weighty and worthy new projects, when wisely matured, gradually overcome the inertia, prejudice and self-

interest which inevitably confront them in their youth.

All of the Boards subscribe to certain general principles and requirements as to purposes, policies and requirements: minima, designed to insure that the examinee shall have been appropriate as a trainee before his special training began, that his special training, itself, shall have been competent; and that, finally, he shall have demonstrated to the Board that he has become a physician whom the Board regards as qualified to practice its clinical specialty expertly, efficiently, ethically.

It is unfortunate, for precision, that the Advisory Board has in its name the expression "for Medical Specialties." That expression is too broad. There are sundry medical specialties, such as anatomy, physiology and the like, for which there exists neither certification purpose nor occasion. Medical licensure is a prime requirement; all of the boards have emphatic clinical background; and the stated expression, plainly, should be "for Clinical Specialties."

One of the norms is the general requirement, "Graduation from a medical school * * * recognized by the Council on Medical Education * * *." However, one, at least, of the boards makes, as a published requirement, graduation from a "university medical school," although its by-laws read simply, "medical school," in harmony with the cited generally adopted norm; and the introduction of this particular foreign body remains unexplained.

The norms have been deliberately and carefully chosen; and, we believe, are broadly sound. However, there is such lack of specificity in various essentials as to make intelligent interpretation impossible. Each board is, apparently, free to make its own interpretation; but even such interpretations are either unobtainable or, if announced, may be too vague to be understandable, or are insular or alien.

This condition is a major fault for such a broad movement. Obviously a prospective examinee or a training clinic or a graduate school or a preceptor-clinician should have access to as reasonably definite and as definitely reasonable specifications as maintain for undergraduate medical education; and these specifications should be reasonably coordinate and harmonious among the requirements of the various Boards.

In brief, we believe that here we have a partly inchoate situation which calls for prompt replacement by logical orderliness.

Voluminous examples of inadequate specifications could be given; but it is appropriate in this necessarily brief article to exemplify only sufficiently for clarity and emphasis.

A general requirement is: "intensive graduate training in anatomy, physiology, pathology and the other basic medical sciences which are necessary to the proper understanding of the disorders and treatment involved in the specialty in question." It is made clear that this training must occur "after the (*required*) internship;" and, from the context, one could, dubiously, conclude that the required time-period for this portion of the training is eighteen months.

We believe that, in practice, unless there here appear clearer specification, each person, or each group, will make his or its own interpretation; that there will be confusion and rapid changes among these separate interpretations; that some interpretations will be sound and sincere; that some will be whimsical; that most will be governed by mere present convenience; and that the general trend would be toward lofty pretense and desuetude.

It does not suffice to make reply that such a matter is incapable of complete specification; and that such specification is undesirably restrictive. Such reply only iterates a truism with which none would disagree. But such reply is inacceptable because it ignores the voids between complete specification and inadequate specification; and between sincerity and evasion. That void is a pathless ocean in which each adept may indeed be his own pilot; but every pilot needs charts and sailing regulations to avoid grounding, collision and substandard insurance.

For constructive examples of reasonable specification, the following items might well become matters of general agreement by the Advisory Board; and, within such general agreement, as indicated, become more specifically defined by each special Board for its own field: (1) Define "intensive" or make substitution or discard the word; (2) Fix partitioned and subpartitioned limits within the ordained total minimal time (Advisory Board, partition limits; special boards, subpartition limits); (3) Agree that such studies, generally, of medical sciences as are merely repetitive of normal undergraduate medical studies are a self-study obligation of the student-physician, himself, and that such repetition constitutes no obligation upon the special instrumentalities utilized in his graduate medical studies; (4) Agree that not only should the

required medical science studies be "applied" in character, but also that they should appear as definite study-entities, in addition to their modernly necessary appearance as casual incidentals in other studies which are essentially clinical rather than medical science study-entities (otherwise the requirement in question becomes pretense as now worded; and the wording should, in sincerity, be altered to disclose the merely incidental character of such studies); (5) Agree, separately in each instance, as to approvable study-methods, especially with regard to cadavera, laboratories and scientific materials and apparatus used specifically for science-study purposes (for example, should the graduate student of surgery be considered to have adequate study of surgical applied anatomy if he have no laboratory cadaver work, but have his anatomic studies consist only of hospital out-patient, bedside, operating room, autopsy and clinical pathology contacts; and, if so, is it reasonable or sincere to term such contacts "intensive graduate training in anatomy"); (6) Agree that, in addition to the approvable clinical study contacts within any major clinical study field, the student-physician shall have conscious, systematic, critical study of the theory and practice of that field as a whole, together with critique of its practical relationships to such other clinical fields as are presently regarded important for such relationships; (7) Agree that this matter of graduate medical education implies serious, extensive and expensive efforts beyond mere clinical contacts, however worthy.

The foregoing constructive examples of how, in our premises, broad specifications are feasible, and may and should become drawn, are consciously incomplete, and intended only to emphasize and clarify our thesis by suitable illustration.

Above, we believe, that with warrant we have spoken not only of an accomplished first stage in the evolution of the clinical certification epoch; but also of an implicitly obvious second stage in that evolution, and the development, nationally and systematically, of connoted educational machinery.

Can one, in reason, think that this machinery shall consist simply of approvable clinics and their clinicians? Certainly not, if the cited general requirements mean what they appear to mean. Those requirements, naturally, make the clinic indispensable to the required training; but, sensibly, they also impose other requirements which involve activities which are not normal to clinics, but which are normal to universities.

This means that the appropriate machinery should be provided through cooperative and coordinate clinic-university efforts. It is true that there are university clinics; but it is also true that their facilities and faculties are already largely engaged by the needs of undergraduate medical education; and that, even though such engagement did not exist, the clinical service volume, herein nationally concerned and implied, far exceeds the combined capacity of university clinics.

What is most needed is widespread conjoint efforts between clinics and universities, without limit as to number and location; limited only by the fitness and the will for services in the premises; and each unit coordinately playing its related normal role.

That does not mean that the clinic so engaged will remain precisely the same in its customs as otherwise it has been; and the university similarly will alter. However, the change at the clinic will become effected readily, whereas the change at the university means large effort and expense to it. We may not here go into details, but may pause long enough to mention two facts. One of these facts is that details for such clinic-university cooperation are available in seasoned and published form. The other fact is that the undergraduate medical educational facilities of universities are inadequate to assume the large additional load implied in this massive movement. Already heavily loaded, they must provide such additional facilities and faculties as shall make their whole effort in basic graduate education in the clinical specialties as serious and conscious an effort as that which undergraduate medical education has modernly become.

There remains, indeed, very much to be done during the cited second stage of the development broadly of clinical service to be rendered wholly by recognized experts.

One of the most nebulous as well as one of the most important and critical of the general requirements is that the practical training locus employed must be "recognized by the Council as competent in the specialty."

The purpose of this requirement is both proper and necessary. However, its implications are so extensive as to call for complete official statements as to criteria; and to call for adoption and publication of procedures for obtaining prompt rulings in special unlisted cases.

Lists of approved special hospital residentships are indeed published annually by the Council. That is right; but is by no means all that is needed.

Implication or assumption that the clinic portion of preparation for special clinical qualification must occur in special hospital residentship, or even that such is the best preparation route, is wrong and should have official disclaimer.

Many, perhaps the majority, of those who presently are our master clinicians have followed other practical training routes, which are economically and educationally sound, and which should remain open and, as found worthy, so be "recognizable" by the Council.

Such routes are usually full or part-time clinical assistantships involving sufficient hospital work on a visiting basis; but, in some cases, involving also the private practices of clinical preceptors. In the same category are also sundry hospitals not presently included in officially recognized lists, but wherein excellent clinical study opportunities do exist.

It is urged that the Council adopt and publish procedures to be followed by those at interest for obtaining from it prompt rulings in individual cases of the stated character. Under such procedures, the presumptive trainee who has such an unlisted clinical study opportunity available to him, should be enabled to ascertain its recognizability before making his decision concerning it.

It is also urged that the Council arrange to compile gradually and to maintain lists of officially recognizable clinical preceptors in our premises, said lists to be in harmony with published criteria; and to comprise both recognized classifications and recognized individuals.

One may well believe that unless the Council and the Boards assume an open and helpful rather than a narrow and rigid attitude, especially in the formative stages, toward the approval of clinical training loci and various types of trainees' associations therewith, that unjust and unnecessary hardships will result for many worthy aspirants for certifications.

This is particularly true for younger physicians, because the moral force or urge upon them will be intense. They will feel that lacking certification they cannot aspire to the better things: in lay and professional standing; in hospital staff posts; in special medical society memberships; in governmental and teaching positions, and so on. The while, many of them, unable to obtain or to afford well known residencies, may nevertheless be situated so as to avail themselves of suitable though presently unlisted ("unrecognized") clinical study facilities.

Commenting further upon the minimal certification requirements, one may note that they provide for a five years' period after internship and before certification examination. Without doubt it is the consensus of opinion of able master-clinicians who have studied the matter that five years is a proper minimum to fix; that of said five years, three years should be devoted to study; and become followed by two years of special clinical practice.

This all seems, upon the surface, to be clear sailing; but there has appeared in certain quarters the will to introduce into the corpus of detailed requirements something which we believe to be foreign to the spirit, purpose and domain of clinical certification, and illogical and harmful.

This foreign body has kinship to the exclusive guild or to the dictatorial trades union. It is an effort to restrict certification to those who have, for specified prior time, exclusively practiced, and who for the future agree to limitation of their clinical services specifically to a single field of certification.

A certificant should indeed have been tested as to his moral and ethical soundness; as to his theoretic and practical qualifications; and as to his due experience; and should conduct himself worthily thereafter; but to go to the extent of using a certification requirement as a weapon to force him to eschew any proper clinical activity on his part is wrong and unwarranted. Such attitude and effort could be assailed in many ways; but let us content ourselves here by saying that there is no reason, for example, why a good surgeon might not be or become qualified for female pelvic work; nor why a good gynecologist might not be or become qualified for male abdominal work; and so on, indefinitely; and we feel sure that none concerned really wishes to achieve the virtual unionization of clinical specialization in any field; or bring about the virtual formation of an American Federation of Clinical Specialists Guild.

It were foolish, even cruel, and it is unnecessary, to ignore the personal economic and civic backgrounds herein involved. The locale of the established metropolitan clinical specialist is no suitable criterion upon which to base certification requirements, which have universal impli-

cations. Each aspirant for certification, each newer certificant, and, generally, each special practitioner must practice his art fittingly for his own locale; and that is his inherent human and professional right, so long as he exercises that right righteously.

Furthermore, there are subspecialties which are entitled to due consideration (examples: thoracic surgery, neurosurgery, bronchoscopy, physiotherapy). Unless and until each oncoming subspecialty waxes to major recognition, as various subspecialties (examples, pediatrics, urology) already have done, the certification of subspecialties must begin with certification for the main specialty of which it is regarded as a part; and later continue with extra certification by the same board. In their planning for an American Board of Surgery, the Committee of Surgeons are taking this point into due consideration.

However, when we consider the matter of long-continued preparatory and probationary periods of exclusive special practices in such cases, the time phase threatens to be prohibitive, unless altered in demand or in definition. For example, how long must the aspirant for eventual neurosurgical certification devote himself exclusively to general surgery before he devotes himself to neurosurgical preparation; and how long exclusively to the latter?

Let us, as stages, summarize and conclude as follows:

(1) Completion of comprehensive plans and organizations for due certification of clinical specialists is now achieved, and terminates the first stage of an epochal advance in the national conditions of clinical practice and clinical service in America.

(2) The second stage is now in progress, and consists: (a) in amplifying, refining and maturing the methods and facilities for educating and qualifying physicians as clinical specialists; (b) and in clarifying, refining and justifying the requirements and procedures for their certification when qualified.

(3) (a) Development of efficient, sufficient and standard clinic-university coordinate efforts will mark the completion of the former division of the second stage; and (b) clear definitions of norms, dissipation of fogs, bridging of hiatus, facilitation of procedures, and elimination of unessentials and inequities will mark the completion of the latter division thereof.

(4) The final stage of this epoch looms and will have become completed when nationally laymen, clinicians, clinics, clinical educators and clinicians' associations shall have made fully kinetic the epoch's vast potentialities. This process, now in its infancy, will probably attain to its maturity within two decades hence, in an era when inexpert clinical service will be taboo.

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DISEASES OF THE NERVOUS SYSTEM CAUSED BY NUTRITIONAL DEFICIENCY*

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Since 1930, when I. S. Wechsler first promulgated the idea that many, or all of the cases of so-called alcoholic polyneuritis might be due to vitamin deficiency, many observers have become interested in this problem which seems to have theoretical as well as practical importance. The idea that neurologic and psychiatric manifestations of alcoholism were immediately caused by the toxic effect of alcohol on the nervous system had been abandoned long ago. The ruling hypothesis had been that of auto-intoxication produced by gastro-intestinal disturbances, the latter being due to chronic alcoholism. However, the notion of gastro-intestinal auto-intoxication seemed somewhat hazy, the toxic substance itself having never been isolated. That nutritional deficiency was a factor, on the other hand, could be shown almost conclusively. M. B. Strauss[21] treated patients suffering from alcoholic polyneuritis with a high vitamin diet which included the amount of alcohol they used to drink formerly. These patients improved just as quickly as those who got no alcohol. Thus, the probability that alcoholic polyneuritis is related to beriberi seems really strong, even if we admit that we are far from any certainty as to the pathogenesis of beriberi itself.

The pathological findings in experimental beriberi are well known. There are degenerations in the peripheral nerves, particularly in their

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most distal parts (H. H. Wollard[32]). The degenerations seem to be identical in alcoholic neuritis and beriberi (Minot, Strauss and Cobb[16]). Beriberi shows definite pathological findings in the spinal cord and in the brain, too. Obviously, degenerations of the central nervous system, which are irreversible, cannot be blamed for curable diseases, as for example, alcoholic psychosis (Schnyder; Koss and Drummond, quot. Kruse and McCollum[12]). In addition, we still do not know what accounts for the pathological processes: whether we have to assume some toxic substance developing somewhere in the organism from vitamin deficiency, or whether there is a metabolic disturbance of the nervous tissue itself, which might require vitamin for its oxidative processes.*

Alcoholic polyneuritis and beriberi can be considered to be identical in their symptoms as well as in their pathogenesis. Furthermore, in all cases of polyneuritis of unknown etiology and with similar symptoms it is justifiable to assume a similar pathogenesis even if there is no supporting evidence of nutritional deficiency, and to start treatment accordingly. However, it is not quite the same in alcoholic psychoses. Of course, we find Korsakoff's syndrome not only in alcoholics, but with other kinds of polyneuritis, and there are cases of Korsakoff's syndrome due to brain injury or to brain tumor; thus, though it be granted that avitaminosis might be one cause, it certainly is not the only cause of Korsakoff's psychosis. On the other hand, delirium tremens and hallucinosis are not found in non-alcoholics, indicating that some specific pathology, perhaps in addition to avitaminosis, is involved in those psychoses. M. B. Strauss[21] believes that "alcohol is poisonous to nerve cells unless there is a sufficient amount of vitamin B or some other dietary factor present to act as an antidote." This may account for some special toxic effect of alcohol made possible by avitaminosis, an acceptable explanation of delirium tremens and hallucinosis; whereas alcoholic polyneuritis would have to be considered as non-specific (absolute or relative) avitaminosis. According to Minot, Strauss and Cobb,[16]

"The consumption of much alcohol may lead to an abnormal increase of daily total metabolism, since a quart of whiskey furnishes at least 2,800 calories so that an unusual demand for vitamin B might develop."

Similar considerations are valid in another problem which has come up along with the recent development of the vitamin pathology: that of the relation of gastro-intestinal trouble, of whatever cause, to syndromes similar to those found in alcoholics. This relation is already known to hold for alcoholism itself. According to M. B. Strauss,[21]

"The high incidence of gastric secretory defects in these patients suggests that poor assimilation of nutriment from the gastro-intestinal tract plays a role in conditioning the deficiency state."

"Many alcoholics," says Wechsler,[29] "do not eat, or if they do eat, do not digest, or if they digest, they do not assimilate. They are food starved and therefore are vitamin starved. So that one is dealing not only with a specific poison but with an additional avitaminosis." (See also Minot, Strauss and Cobb[16].)

O. C. Perkins[18] found, among 42 cases of multiple neuritis with chronic alcoholism, 31 with achlorhydria, 8 with hypochlorhydria, 30 with intestinal disturbances. He found similar percentages in his cases of alcoholic wet brain with polyneuritis and delirium tremens with polyneuritis. There are quite similar reports of gastro-intestinal trouble, particularly achlorhydria, in pellagra (Eusterman and O'Leary). In addition, there are early as well as recent observations on polyneuritis following postoperative vomiting, occlusion of the common bile duct with cholemia, operations on the gastro-intestinal tract (for example, colostomy, ileostomy, gastro-enterostomy), ulcerative colitis, cancer, and benign lesions such as benign achlorhydria (Winans and Perry; Christopher, Paskind and Snorf; J. M. Barnes; Urmy, Ragle, Allen and Jones; C. M. Jones; O. C. Perkins). Again, our material as well as that of other observers is able to support that interpretation. The fact that peripheral neuritis and polyneuritis with specific etiology are found mostly after those intoxications (arsenic, lead) and infections (typhoid fever, dysentery) which also severely injure the gastro-intestinal tract (see Wechsler), certainly suggests a unitarian point of view which would bring these conditions into some close relationship with avitaminotic processes. The relation between gastro-intestinal disturbance, nervous disease, and avitaminosis may have two different interpretations:

*According to Kinnersley and Peters (quot. Winans and Perry,[31] Kruse and McCollum[12]), polyneuritis of vitamin B_1 deficiency is associated with a localized accumulation of lactic acid in the brain, and Meikeljohn[14] found that concentrates of that vitamin were capable of remedying this condition. "The lesion results in a lowered oxygen uptake of the minced brain in the presence of lactate. The addition of vitamin B_1 concentrate to the minced brain largely restores the lowered uptake . . . The lesion in vitamin B_1 deficient brain affects an oxidase system that is associated with lactate but is not concerned with the removal of lactate itself . . . "

on the one hand, it may be that the serious diminution of absorption due to gastro-intestinal disease involves absorption of vitamins, too, and that vitamin deficiency brings on the nervous disease (G. R. Cowgill). In accordance with this view, Minot (quot. Cowgill) believes that achlorhydria, which is often found in beriberi and in chronic alcoholism, may prevent the utilization of B vitamins.

The second interpretation would be that we are dealing in all cases with starving processes, which in beriberi, pellagra, and so on, are caused by lack of those vitamins necessary for utilization of food, whereas in cases of severe gastro-intestinal trouble, malignancy, and other consumptive processes, cachexia and nervous disturbance are directly caused by lack of foodstuff or inability to digest it. According to Winans and Perry,

"Any prolonged illness accompanied by reduced food consumption might be expected to produce the disease." (See also I. S. Wechsler.)

It may be pointed out that gastric ulcer, even when associated with hyperchlorhydria, may indirectly cause avitaminosis due to the therapeutic restriction of diet.

This probably holds for diabetes, too, particularly in cases in which a very strict diet is observed (see Wohl, quot. G. C. Shattuck). The fact that starvation produces pathological changes in the peripheral nerves quite similar to those of vitamin deficiency has been proven by experiment. Besides, there is no doubt that every deficiency of vitamins of the B group will cause gastro-intestinal disturbance. Withdrawal of vitamin B reduces motor activity of the gastro-intestinal tract (L. Gross, B. A. Plummer; Rose Stucky and Cowgill; all quoted from T. T. Mackie). Chronic ulcerative colitis may be caused by avitaminosis (Dickson, Larimore, quot. Mackie). Experiments made by Webster and Armour prove that the vitamins contained in yeast are necessary for the normal secretory activity of the gastric mucosa. If now we take it for granted that absorption of vitamins is checked by gastro-intestinal disturbance we find a vicious circle, either of the factors being responsible for nervous trouble (see also Wechsler).

At this point, pregnancy, as one of the conditions frequently predisposing to polyneuritis and acute psychoses, deserves mention. There is a well-defined series of symptoms occurring in pregnancy, including achlorhydria, hyperemesis, anemia, polyneuritis, and mental disturbance. Each of these symptoms may occur alone or combined with any of the others. The question of their relationship, whether the gastro-intestinal disturbance, being toxic in origin, accounts for some or all of the other symptoms, or whether all of them are coordinated sequelae of vitamin deficiency, has not yet been answered. At any rate, avitaminosis seems to be one of the connecting links in the chain (Strauss and Berkwitz, Strauss and Castle quot. Winans and Perry; Berkwitz and Lufkin quot. Strauss and McDonald; Wechsler, Shattuck, Perkins).

But one must beware of undue simplification. Referring all nervous symptoms to nonspecific gastro-intestinal trouble would fail to account for special characteristics differentiating, for example, lead polyneuritis from arsenic polyneuritis, and both of them from other types; it would fail to explain the fact that a combination of localized cerebral, spinal, and peripheral lesions in one case, otherwise rare, is frequent after typhoid fever. Again, we must assume certain specific factors in addition to the metabolic one. The latter seems to be alone responsible in cases of polyneuritis, with or without acute psychosis, due to nonspecific gastro-enteritis, carcinoma of the stomach, gallbladder trouble, or gastro-enterostomy. According to our experience, this probably holds also for postdysenteric polyneuritis, which does not exhibit any peculiar symptoms, but is entirely similar to beriberi.

It must be admitted that our conclusions concerning the avitaminotic origin of any disease whatever are not altogether well founded as long as we have only the case history and the therapeutic test to rely upon, even if the latter turns out to be conclusive as in the cases of Vorhaus, Williams and Waterman. What is needed is a specific chemical or biological test for avitaminosis. As far as avitaminosis B_1 is concerned, a Japanese observer (Kimura) recently published important findings: lactating mothers with a negative Arakawa reaction of their milk, the negative reaction being characteristic of vitamin-deficient milk, showed a considerable increase of their blood platelet count, and this could be reduced to normal by vitamin B_1 administration. However, in a series of observations of our own, which is admitted to be small, we did not find an increase of blood platelets in a single case, the cases examined being of true beriberi and alcoholic beriberi. Furthermore, these cases improved markedly after vitamin administration, without any change of their blood platelet count, which had always been normal.

The problem of peculiar functions of different vitamins, which seemed to be rather simple about ten years ago when vitamin B and antineuritic vitamin were still considered the same, has become rather complex at the present time. The vitamin B group, according to present knowledge, contains at least six different vitamins. The structural formula of vitamin B_1, which is still considered specific for beriberi prevention, has recently been established, only one insignificant detail of the formula being still in doubt. A crystalline vitamin B_1 has been produced, and used in clinical practice. The structure of vitamin B_2 is entirely known; it has been recognized as lactoflavin (d-ribo-flavin) (Von Euler). Meanwhile, its anti-pellagric effect has become doubtful. According to recent findings, the "blacktongue" of dogs, which is certainly due to vitamin B_2 deficiency, has nothing to do with human pellagra, the latter being caused by deficiency of some other B-vitamin, possibly B_6.* Incidentally, the distinction between "true" pellagra and "alcoholic pseudo-pellagra" is no longer maintained. According to Spies and deWolf, over 90 per cent of the patients with pellagra developed the disease following severe alcoholism. The experiment tried in alcoholic polyneuritis (see above) gave positive results also in pellagra: patients will recover following administration of vitamin regardless of whether they keep on drinking or not (Spies and deWolf).

Besides the vitamin B group, the fat-soluble vitamin A seems to be involved in diseases of the nervous system, according to Mellanby's well-founded experimental findings. It seems to us particularly important that Mellanby found spinal cord changes in experimental vitamin A deficiency. Avitaminotic spinal disease had formerly been described by Hart, Miller and McCollum (quot. Mellanby), but lately has not received much attention, with the exception of combined tract degeneration, which now, in association with pernicious anemia and achlorhydria, is considered to be avitaminosis due to lack of the "liver-factor."†

However, there seems to be some evidence, particularly in our own material, that not only combined tract degeneration, but also non-specific transverse myelitis may be ranged among the deficiency diseases. In addition, we saw several cases of combined tract degeneration without either pernicious anemia or achlorhydria and one case of subacute spastic spinal paralysis, all of which possibly may belong here.

August Meyer believes that funicular myelitis is not uncommon in alcoholism and assumes a close relation of beriberi and pellagra to that condition (see also Gildea, Kattwinkel and Castle, B. R. Tucker, Orton and Bender). Tucker reports pathological findings in the spinal cord in cases of pellagra. Whether or not Mellanby's experimental findings concerning vitamin A deficiency have anything to do with clinical experiences concerning spinal disease, we do not know. The pathological findings reported by Winckelsmann and by Singer and Pollock (quot. Orton and Bender), concerning spinal cord pathology in pellagra and chronic alcoholism suggest the hypothesis that B group avitaminoses, too, seem to have some affinity for the spinal cord. On the other hand, Mellanby believes that "vitamin A or carotene plays a part in the prevention of pellagra or at least of its nervous manifestations." Thus it seems that the former notion of peculiar action of each vitamin has been somewhat unsettled.

"It has become obvious that, though specific in many respects, the effects of deficiency of the various vitamins show overlapping, and are not confined exclusively to the production of a definite and characteristic symptomatology" (E. Browning).

Even the antiscorbutic vitamin C has to be considered. Nonne (quot. Minot, Strauss and Cobb) pointed out that an analogy might be drawn between the vascular lesions of scurvy and those found in polio-encephalitis superior due to chronic alcoholism.

In addition, the strict borderline between vitamins and hormones is about to disappear. Recent findings stress the similarity, perhaps even identity, of ovarian hormone (estrogenic substance) and vitamins A and E (E. Browning). Vitamin B_1 seems to be closely related to the thyroid (R. L. Haden). According to Himwich, Goldfarb and Cowgill, loss of appetite and weight in dogs, caused by thyroid administration, can be remedied by vitamin B. Anorexia due to Cowgill's artificial diet occurs earlier when thyroid is added.

"Those results harmonize with the theory of Plimmer and Cowgill and Klotz, that the amount of vitamin B required by the organism is determined chiefly by its caloric requirement" (see also G. R. Cowgill).

It may thus sooner or later develop that only one kind of catalytic ferment is required for

*Chick and her co-workers and Gyorgy are of the opinion that vitamin B_6 together with flavin (B_2) forms a combination having the total biological action formerly attributed to B_2 (Von Euler).

†Non-pernicious anemia can be caused by vitamin A and B deficiency (Funk, Whitehead and Barlow, quot. Browning); the same holds for anemia of pregnancy (Wills and Mehta, quot. Browning). Mellanby suggests that the liver factor, which may or may not be identical with vitamin A, prevents and benefits ergotism, ergot being contained not only in *Claviceps purpurea* but in wheat germ or embryo as well.

metabolism: these catalysts are hormones while working within the same organism, be it plant or animal, which produces them, and appear as vitamins when this organism is used as a foodstuff; thus other organisms are supplied with catalysts not at all or insufficiently represented among the latter's own hormones.

* * *

Our own material consists of 47 cases of various localization and etiology, all of which have this in common: that a metabolic disturbance along the line of avitaminosis is either certain or strongly probable. The cases were diagnosed as shown in *Table 1.*

Table 1

CLASSIFICATION OF CASES

Acute psychosis	4
Acute psychosis with polyneuritis	1
Polyneuritis	6
Beriberi	2
Pellagra psychosis	1
Pellagra polyneuritis	1
Puerperal polyneuritis	1
Cachectic psychosis	1
Alcoholic hallucinosis	4
Alcoholic Korsakoff's syndrome	1
Alcoholic polyneuritis	9
Alcoholic delirium and polyneuritis	1
Alcoholic hallucinosis and polyneuritis	1
Alcoholic delirium, Korsakoff's syndrome and polyneuritis	1
Alcoholic Korsakoff's syndrome, polyneuritis and pellagra	1
Alcoholic hallucinosis, polyneuritis and pellagra	1
Alcoholic transverse myelitis	2
Transverse myelitis	4
Combined tract degeneration	3
Transverse myelitis and Korsakoff's syndrome	1
Combined tract and anterior horn degeneration	1
Total	47

As can be seen from the table, 21 of the cases are considered alcoholic in origin, all but two presenting the usual alcoholic symptoms of polyneuritis, delirium, hallucinosis and Korsakoff's syndrome in various combinations, in two cases together with pellagral dermatitis. We added two cases of transverse myelitis, not because we are sure of their alcoholic origin, but because that etiology certainly ought to be considered. Besides by the therapeutic test, this hypothesis is supported by the fact that among the four cases of transverse myelitis there was one with Korsakoff's syndrome, this case apparently being of gastro-intestinal origin. The question of how much we are entitled to include transverse myelitis in this series will be discussed later.

Concerning the etiology of the remaining 26 non-alcoholic cases, *Table 2* may be consulted.

Table 2

ETIOLOGY

	Gastro-intestinal	Starvation	Cachexia	Puerperal	Acute Infection	Unknown	Total
Acute psychosis	1		1		2	1	5
Acute psychosis with polyneuritis	1						1
Polyneuritis	2			1	1	3	7
Beriberi		2					2
Pellagra psychosis		1					1
Pellagra polyneuritis	1						1
Transverse myelitis						3	3
Combined tract degeneration						3	3
Combined tract and anterior horn degeneration						1	1
Transverse myelitis with Korsakoff's syndrome	1						1
Total	7	3	1	1	3	11	26

It will be seen that the largest number, as might have been expected, is that of cases without any known etiology (11 cases). The conclusion that metabolic disturbance might account for them is primarily based on their likeness in symptomatology to cases of better known etiology, such as alcoholism and beriberi, and only secondarily on the therapeutic test. We know, of course, that neither of those inferences is sure; however, we do not claim theoretical certainty which, as has been pointed out above, could only be obtained by a specific chemical or biological test; we merely want to use the conclusion as a heuristic hypothesis and for purposes of practical therapy. The decision to include transverse myelitis in this series was made by the fact that, in several cases of that lesion, the results of vitamin administration were surprisingly similar to one another and to the results in cases of combined tract disease: almost regularly, the beginning of treatment was followed by sudden improvement of all neurological symptoms which, up until then, had been constant or even progressive for months or years; but the improvement would stop after one or two weeks and no further change in the patient's condition could be effected. This is exactly what might be expected in a spinal disease of metabolic origin: the vitamin administration brings about prompt repair of those toxic lesions of the nervous tissue which are still reversible, whereas total degenerations of fibers and cells due to prolonged toxic action could not, of course, be repaired. Thus, the final outcome of cases of acute or subacute myelitis, whether

transverse myelitis, combined tract disease, or, as in one of our cases, combination of the latter with anterior horn involvement, would be about the same as the ultimate stage of anterior poliomyelitis, when all structures still amenable to repair have been healed and the residual palsies are due to irreversible destruction of ganglion cells.

The working hypothesis we have in mind could not be upheld unless we enlarged the conception of avitaminosis even beyond the boundaries already stretched to include alcoholic and gastrointestinal etiology. For that purpose, we want to introduce the name of *neurometabolic crisis* as a substitute for avitaminosis. This term means: (1) that we are dealing with processes going on in the nervous system, with various localizations; (2) that metabolic derangement is one of the pathogenic factors, perhaps the most important one; (3) that the process has, primarily, the character of a crisis, that is, of an acute or subacute disturbance which may end either in recovery or in an irreversible degenerative process. The latter may be due to the continuation of the metabolic derangement itself or to some synergic factor of another kind, such as constitutional organic inferiority, or intercurrent infection. The metabolic derangement itself may be caused by starvation, by unbalanced diet, perhaps an excess of carbohydrates in the food, by deficiency of absorption due to gastrointestinal disease, or by an absolute or relative lack or surplus of catalysts required for nervous metabolism, the catalysts being supplied by vitamins on one hand, or by endogenous hormones on the other. Since the metabolic derangement we have in mind is mostly confined to the nervous system, it certainly cannot be demonstrated by the general metabolic tests available such as basal metabolic rate, carbon dioxide combining-power, nonprotein nitrogen, and so on. We probably are dealing with either cellular starvation or cellular asphyxia of the nervous tissue, which may or may not be accompanied by a general metabolic disturbance.

Among the apparent causes quoted in our table, acute infection figures in only three out of 47 cases, which certainly is a very small number considering the fact that infection has been believed to be one of the most important causes of polyneuritis, acute psychoses, and transverse myelitis. It seems that infection has been accused mostly when there was no evidence of any other etiology. Some bad teeth, chronically infected tonsils, sinuses, or appendices were at hand in most cases. In our three cases, we firmly believe that infection had something to do with the disease, but only in the sense which holds for "infectious" beriberi, too. The increased basal metabolism in fever may bring on a relative deficiency of catalysts, vitamins or hormones, whenever the supply of catalysts has been rather low, temporarily or continually just above the concentration required for normal metabolism. Once the neuro-metabolic crisis is started, a vicious circle develops with gastrointestinal disturbances caused by the deficiency and, in turn, increasing the deficiency by lessening absorption.

It deserves to be mentioned that among the 25 cases of polyneuritis with or without cerebral involvement, there is not even one due to any other poison than alcohol. And yet this series includes all the cases of polyneuritis we saw in a period of one and a half years with the sole exception of some old cases of "jake" paralysis which did not seem to us to belong here. It seems from this that mineral poisoning as a frequent cause of polyneuritis has been overestimated, if we leave out obvious industrial poisoning with lead or arsenic. In a series of observations on polyneuritis and neuritis published in Europe during the World War, I still believed that unrecognized cases of poisoning, particularly with lead, might account for the tremendous increase of incidence of peripheral nerve involvement at that time. Today, I am almost convinced that the increase was due simply to starvation, this being the most probable cause in view of the very poor food supply of the Austrian army from 1916 on. It is an interesting fact from the standpoint of war psychology, that nobody at that time, including myself, had any suspicion of that etiology.

An analysis of the gastric contents has been done, for technical reasons, in only 11 of our 47 cases. The acidity was found normal in three cases (two cases of alcoholic polyneuritis with psychoses, and one of combined tract degeneration) increased in two cases of transverse myelitis, and decreased or almost absent in 6 cases (3 cases of pellagra polyneuritis, 2 of them combined with psychoses and one of these alcoholic in origin; one each of alcoholic polyneuritis, alcoholic hallucinosis, and transverse myelitis with Korsakoff's syndrome). The incidence of hypochlorhydria in 6 out of 11 cases is not quite so high as might have been expected according to the experiences of other observers. However, it is still high enough to justify the

belief that the function of the gastric glands plays an important role, either as a coordinated symptom or as a causal factor, or as both, in the neuro-metabolic crisis. It may be remarked that all three of our pellagra cases examined for their gastric secretion were hypo-acid or anacid, and that the only cases in which hyperacidity was found were two cases of transverse myelitis out of three examined. Of course, the number of cases is by far too small to permit the drawing of any conclusions whatever.

Finally, we wish to give in *Table 3* the results of treatment and the final outcome in those 32 cases in which observation was long enough to allow judging. Omitting three cases which died from intercurrent diseases, there were 22 cases treated with high vitamin diet, whereas 6 received ordinary diet. Two of these 6 remained unimproved; but only one out of 22 in the high-vitamin-diet group went without improvement. In addition, it must be noted that the regular hospital diet, unsupplemented, is probably richer in vitamin than the average diet of the Southern farmers and laborers who come to the Charity Hospital of Louisiana. This may partly account for the improvement shown in the 4 cases with regular diet.

Table 3

TREATMENT

	Regular Diet Improved	Regular Diet Not Improved	High Vitamin Diet Improved	High Vitamin Diet Not Improved	Deaths	Total
Acute psychosis	1		1			2
Acute psychosis with polyneuritis			1			1
Polyneuritis			4			4
Beriberi			2			2
Pellagra psychosis					1	1
Pellagra polyneuritis	1					1
Puerperal polyneuritis			1			1
Alcoholic hallucinosis			2			2
Alcoholic Korsakoff's syndrome			1			1
Alcoholic polyneuritis	1	2	2	1		6
Alcoholic hallucinosis with polyneuritis			1			1
Alcoholic Delirium, Korsakoff, polyneuritis			1			1
Alcoholic Korsakoff, polyneuritis, pellagra			1			1
Alcoholic hallucinosis, polyneuritis, pellagra			1			1
Transverse myelitis			2		1	3
Combined tract degeneration	1		1			2
Combined tract degeneration, ant. horn degener'n			1			1
Transverse myelitis, Korsakoff's syndrome					1	1
Total	4	2	22	1	3	32

The high vitamin diet given in our hospital included all kinds of vitamins, without laying particular stress on the non-fat-soluble B group. The patients were given fresh vegetables, fruit, fruit juices, particularly lemon and grapefruit juice, tomato juice, codliver oil, yeast, and liver extract. We do not believe that the special values of different vitamins are at present so well established as to justify any distinction in the therapeutic diet.

As far as the influence on various symptoms is concerned, we were particularly struck by the fact that psychoses seemed to react to the dietetic regime by far faster than neurological symptoms. The typical progress in our cases of polyneuritis with mental disease, with or without alcoholic background, was that the psychosis disappeared within a few days, whereas the polyneuritis required weeks and even months of slow improvement before the patient could leave the hospital cured or considerably improved. Of special interest seemed to us the case of a 32-year-old Negro, who came to the hospital in a state of alcoholic hallucinosis. The first examination elicited no neurologic signs whatever. In particular his tendon reflexes were entirely normal. He recovered entirely from his psychosis, on a high vitamin diet, after three days. On the same day, when he became rational again, he complained of severe pains in his legs and weakness. At this time neurologic examination revealed severe ataxia of both lower extremities, tenderness of calves, and entire loss of both patellar and Achilles reflexes. The complete picture of an ataxic polyneuritis had developed acutely, and it took four weeks of treatment to bring about a considerable improvement, whereby the patient was able to leave the hospital walking, but still without his reflexes. It seems that in this case the vitamin treatment was sufficient to check the metabolic crisis in the brain, but came too late to prevent it in the peripheral nerves, which were much more severely affected.

Omitting all theoretical discussion of the avitaminotic origin of the conditions considered, we do believe that the therapeutic results of other observers, as well as ours, are encouraging enough to justify recommendation of therapeutic high vitamin diet in all cases of (1) non-specific or alcoholic neuritis and polyneuritis, (2) com-

bined tract degeneration (funicular myelitis) and transverse myelitis, (3) acute and subacute "toxic" psychoses, such as states of confusion (amentia), delirium tremens, hallucinosis and Korsakoff's syndrome. In addition, we want to emphasize that even in cases with a specific infectious or toxic (non-alcoholic) etiology, high vitamin diet may be useful as an accessory treatment, since we do not yet know how far the specific factor alone is responsible for the disease. Therefore, though we cannot report experiences of our own as yet, we recommend high vitamin diet also in cases of polyneuritis from lead or arsenic poisoning, postdiphtheritic polyneuritis, luetic transverse myelitis, and even in certain cases of cerebral lues.

SUMMARY

Forty-seven cases of peripheral, spinal and cerebral diseases are reported in which metabolic deficiency as a pathogenic factor is either certain or highly probable. The concept of "avitaminosis" is enlarged to the idea of "neurometabolic crisis" which may be brought on not only by vitamin deficiency, but also by other factors involved in metabolism.

Finally, high vitamin diet is recommended to be used in all cases of a similar nature, even with unknown etiology, and as an accessory treatment in cases with specific etiology.

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DISCUSSION (*Abstract*)

Dr. Titus H. Harris, Galveston, Tex.—Dr. Wexberg has presented an excellent discussion of the part played by nutritional deficiency in the production of certain neuropsychiatric disorders. He reviews the literature and discusses the attitude of present day investigators regarding the role of avitaminosis as the direct or contributory factor in multiple peripheral neuritis, in some types of myelitis and certain mental disorders. By pointing out the similarity of alcoholic polyneuritis to the neuritis of beriberi, he re-emphasizes the changing ideas concerning the toxicity of alcohol. During the past few years, I have treated several cases of alcoholic polyneuritis by giving substances high in vitamin B content. The patients responded to this therapy in an amazingly rapid fashion, especially when compared to previous cases of this type that were not treated in this manner. Although Dr. Wexberg questions the part played by vitamin deficiency in producing alcoholic mental disorders, there is room for further investigation and thought along this line.

Vitamin B is apparently the chief offender, for the gastro-intestinal tract is also affected by its absence, but the other vitamins, particularly A, may be associated with spinal cord changes. The cases reported of postero-lateral sclerosis and transverse myelitis are of especial interest to me. Three years ago I had a patient with symptoms of a transverse lesion. At first he was thought of as having a spinal cord tumor, but later showed a blood picture resembling pernicious anemia. He was treated for this disease with liver extract and showed rapid improvement, and though he was unable to walk when admitted to the hospital, when discharged, he walked with little difficulty. This patient was not seen again until a few weeks ago, and he is still in good condition, having done hard farm work during the intervening years. The point of interest about this patient is that he left off his liver

extract soon after leaving the hospital, and he has taken no treatment since.

Is it possible that the poor absorption of vitamin by the gastro-intestinal tract produced the myelitis? In keeping with Dr. Wexberg's explanation of the "vicious cycle," a deficient intake of vitamins over a period of years could have produced this altered gastro-intestinal function. Replacing the vitamins corrected the difficulty to the extent that the stomach and intestines in this case were able to continue proper absorption.

Another patient with undoubted pernicious anemia developed an acute transverse lesion after leaving off the liver extract. This cleared up completely after resuming the treatment and the patient has remained free from symptoms up to the present time, a period of more than two years.

Both of these cases support Dr. Wexberg's opinion that acute and subacute myelitis may result from vitamin deficiency.

I should like to mention the occurrence of pellagrous dermatitis and accompanying gastro-intestinal symptoms in patients who refuse to take sufficient food. Chronic depressions with retardation, schizophrenics with delusions, psychoneuroses with vomiting, and various other forms of mental disorder are examples of patients showing this meager food intake. These early cases show no neurological involvement and the symptoms and signs clear up rapidly with administration of liver extract intramuscularly, brewers' yeast by mouth, and the ordinary "house diet" of the hospital.

Dr. Wexberg reports several cases of acute psychoses which, I presume, were of the organic type. For some time I have been seeing cases of toxic or organic psychoses where complete study revealed vitamin deficiency as the probable cause. These cases presented a picture of confusion with defective mental grasp and capacity, and they were chiefly in middle-aged or elderly people. Physically, all were greatly undernourished and many had red, atrophic tongues with a bilateral dermatitis over elbows, hands or the dorsum of the feet. There were a number of cases that did not show changes in the skin or mucous membranes. In other words, the classical picture of pellagra was rare. I feel that a low grade degenerative type of encephalitis results from inadequate vitamin intake with the earliest symptoms resulting from the edema accompanying any inflammatory change. Such a viewpoint would explain the complete disappearance of all sensorium changes in those cases of short duration that receive vitamin therapy. Where the symptoms have been present for years, the administration of vitamins is ineffectual as permanent degenerative changes have occurred.

In May of this year, a 78-year-old minister was brought to the hospital because of irrational talk and behavior. Four months prior to admission he was forced to give up his ministerial duties much against his own wishes. The end seemed to have come for him and he sat around home brooding and complaining. He gradually became more despondent. Difficulty in sleeping occurred. The patient would eat only when urged; even then only small quantities. During the month prior to admission he began to show signs of memory loss. This increased gradually until ten days before he was admitted, when he began to talk irrationally, was disoriented for place and person, and became fearful of being killed. Such behavior continued until his admission. During the period of confusion, his food intake was almost nil. Physical examination revealed a poor state of general nutrition, a brownish pigmentation over the dorsum of both hands, blood pressure of 160 systolic and 94 diastolic, advanced retinal sclerosis, and a compensating arteriosclerotic heart disease. The mental status was characterized by extreme confusion with delirium. Although too weak to stand, he would throw himself about, shout or mutter constantly, revealed apprehension, and talked of being mutilated, killed or of being infected with a terrible disease. He was incontinent, had to be tube fed. The sensorium revealed profound changes.

Sedatives were used for rest, fluids were given under the skin for the first few hours; then concentrated, nutritious, high vitamin fluid feedings were given by nasal tube. Concentrated liver extract in 3 c. c. doses was given every other day. By the eighth day he was taking some nourishment by mouth. At the end of two weeks he was cooperating fairly well. In three weeks he was showing only transient periods of disorientation for time, and some loss of recent memory. At the end of the month he was placed in the general hospital with most of the responsibilities of caring for himself. He was discharged a month later showing only the symptoms of a mild depression. I have seen him several times since, and his sensorium has remained clear.

The patient was investigated completely for all possible etiological factors. The type of reaction, which was an acute or organic or toxic psychosis, did not belong to the senile or arteriosclerotic groups. The skin manifestations pointed to vitamin deficiency, and the history of refusal of food supported the idea. This conception was strengthened further by the recovery of the patient, who received no specific therapy except general care and a high vitamin diet.

DIETARY DEFICIENCIES AS A CAUSE OF ANEMIA*

By PAUL W. CLOUGH, M.D.
Baltimore, Maryland

The anemias are commonly classified on the basis of the morphology of the red cells into three main groups: the hypochromic microcytic anemias, the macrocytic (hyperchromic) anemias, and the normochromic (usually normocytic) anemias. These designations are useful because they are simple descriptive terms and because a majority of cases in each group have other fundamental characteristics in common.

The hypochromic anemias include chronic posthemorrhagic anemias, hookworm anemia, certain nutritional anemias, chlorosis and idiopathic hypochromic anemia. In all of them a deficiency of available iron is associated with the

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anemia, and all are benefited by the administration of iron, and not of liver extract.

The macrocytic anemias include pernicious anemia, fish tapeworm anemia, and some of the cases of anemia which occur in sprue, idiopathic steatorrhea, pregnancy, and in advanced stages of cirrhosis of the liver. In this group the factor common to many of them seems also to be a deficiency, not of iron but of the anti-anemic substance found in liver. They are further characterized by their favorable response to liver preparations and not to iron.

The normochromic anemias are a heterogeneous group secondary to various systemic diseases and are apparently due to a depression of hemopoiesis rather than a lack of any specific substance.

Disregarding this group and also the relatively rare hemolytic and myelophthisic anemias, there remain two large groups in which the cause is to be found in a deficiency of one or more essential substances. Some of these enter directly into the composition of the cell as building stones, while others act indirectly, by influencing in some way the processes of cell formation and maturation.

The red cell is made up of hemoglobin imbedded in a relatively small bulk of stroma. The hemoglobin is composed of iron united with a protoporphyrin nucleus which is combined with a complex protein molecule, globin.

The most common anemias are due to a deficiency in the "building stone" iron. This may result from an inadequate diet, from failure to absorb or utilize iron, or from loss by repeated hemorrhages. A quantity adequate normally may be deficient when the need for iron is great, as in pregnancy. An absolute deficiency of iron in the diet is relatively rare in adults, but it is common in infants and small children who are kept for a long time on a diet composed largely of milk. At birth the infant, even of an anemic mother, nearly always shows a high red cell count and hemoglobin (120 per cent or more), which is attributed to the stimulus of the low oxygen tension in the placental circulation. After birth the surplus cells are gradually disposed of and there is a physiological fall in hemoglobin during the first three months which cannot be prevented by diet or medication. The iron thus liberated is retained in the liver and added to the store which is normally accumulated there during the last few months of fetal life. This store usually supplies the needs of the infant for the next two or three months, but if a milk diet is continued there is a second fall in hemoglobin. This can be prevented by the administration of iron or by the addition to the diet of foods rich in iron. The great frequency of this anemia in the poorer classes of the large cities is shown by the observations of Mackay[1] in Aberdeen, who found a significant degree of anemia in 50 per cent of the infants of that age. The practical significance of this anemia is shown by the fact that the total morbidity among anemic infants is about twice as great as among those with normal blood.

The amount of iron required by older children and male adults is comparatively small. Davidson[2] has shown that under favorable conditions equilibrium can be established on a daily intake of 6 mg., which is usually provided by a general diet. This, however, does not provide any reserve margin of safety. Any extra strain such as may be imposed by an infection will precipitate an anemia. Women require more iron. Davidson found that 50 per cent of the women of the poorer classes showed an anemia, which was severe in 15 per cent. A study of their diet showed that the average daily iron intake was only 10 mg. and in many cases was 6 mg. or less. To provide a reasonable margin of safety the intake should be at least 15 to 20 mg. a day, and this can be provided if some care is used in the selection of foods.

Chlorosis results from an excessively low intake of iron, as is shown by some old observations of Stockman,[3] who estimated that the daily intake in 3 cases was from 1 to 3 mg. The rarity of such extreme deficiencies in modern diets adequately explains the practical disappearance of the disease.

A virtual deficiency in iron may be caused by defective absorption in the gastro-intestinal tract. This is particularly apt to occur in conditions such as sprue, idiopathic steatorrhea and chronic ulcerative colitis.

Absorption of iron is also reduced if there is no free hydrochloric acid in the gastric juice, as for example, in idiopathic hypochromic anemia. All these factors combine to bring about a grave depletion of the iron stores. This is manifested not only by the markedly hypochromic character of the anemia, but by changes in other tissues also, by the dry, lusterless skin and hair, the thin, brittle, spoon-shaped nails, and possibly by the atrophy of the buccal mu-

cous membranes. The administration of large doses of iron usually forces the absorption of sufficient iron to supply the need, and rapidly relieves these abnormalities as well as the anemia.

Only soluble dializable iron salts can be absorbed from the intestinal tract. Any iron occurring in the food in organic combinations is useless until it is split off and reduced to a simple soluble salt by the digestive juices. These processes are favored by an acid medium and are seriously disturbed if achlorhydria is present.

The inorganic iron in the food is present chiefly in the form of ferric salts. There is evidence that in the contents of the gastro-intestinal tract these salts tend to form combinations with the proteins and sugars present which interfere with their absorption. It is probable that iron is absorbed largely if not entirely as ferrous salts.[4] In the normal acid stomach contents conditions are favorable for the reduction of ferric salts to the absorbable ferrous salts. In an neutral or alkaline medium this reduction process is interfered with, and most of the iron escapes absorption. Clinical studies indicate that as judged by the size of the dose required, ferrous salts are about six to ten times as effective as ferric salts.[5] The ferric ammonium citrate which has been so extensively used is therefore one of the poorest preparations that could have been chosen for patients with achylia, although even it is effective if sufficiently large doses are administered.

Among the other building stones of hemoglobin is globin or its constituent amino acids. There is little positive evidence as to the part which these actually play in the production of anemia. That the lack of substances of some sort other than iron may be significant is indicated by the experiments of Whipple and his associates on dogs which were kept on a diet poor in iron and which were kept anemic by repeated bleedings. When whole liver or kidney was added to the diet, together with medicinal iron, there was an accelerated formation of hemoglobin which could not be accounted for by their iron content alone. In the treatment of hypochromic anemia it is therefore important not merely to supply iron, but to give a well balanced diet, including meat and liver, in order to insure an adequate supply of all the raw materials for hemoglobin formation.

To secure normal blood regeneration, certain substances are required which do not enter into the composition of the cells themselves, and presumably act as catalysts or possibly as hormones. One of these is copper. The need of copper was first shown by Hart, Steenbock and Elvehjem[6] in the case of rats made anemic by a milk diet, and their findings have been amply confirmed. A grave nutritional anemia in cattle due to a deficiency in both iron and copper has been described in the case of herds pastured on ranges the soil of which is extremely deficient in these elements. Such "salt sick" animals can be cured by feeding them iron-copper mixtures, but not iron alone. The amount of copper required is so small, however, for man, about 2 mg. a day, that even the poorest diet supplies it, and the therapeutic administration of copper as a supplement to iron is probably never necessary except possibly in young children.

In the macrocytic anemias there is a lack of some substance which normally is formed in the stomach and stored (and possibly elaborated) in the liver. Lack of this substance leads to retarded and defective development of the red cells, but apparently does not interfere with hemoglobin formation. If Castle's hypothesis is valid, as it probably is, despite some recent criticism, this deficiency may be caused either by the failure of the stomach to secrete the intrinsic factor, or by a deficiency in the diet of the extrinsic factor, a substance which is intimately associated with, but probably not identical with, vitamin B_2.

A primary deficiency in the extrinsic factor is rare in Europe and America, but it is not uncommon in the tropics in patients who subsist largely on milled cereals. However, even when the dietary is adequate, serious intestinal disturbances which lessen absorption such as sprue and chronic colitis may cause an actual deficiency in this anti-anemic principle.

By the administration of "vegex" or "marmite," which are rich in extrinsic factor, it is often possible to secure the production and absorption of sufficient anti-anemic principle to cure the anemia.

The resemblance of the degenerative changes in the nervous system in pernicious anemia to those in pellagra, their relative refractoriness to treatment and other facts have suggested that these degenerations may be due to some deficiency other than that which causes the anemia. Experimentally lesions in the nervous system of animals somewhat resembling those seen in pernicious anemia have been reported in deficiency of vitamin A,[8] of the B complex,[9] and less clearly B_2.[10] However, the administration of these vitamins does not affect the condition

in pernicious anemia, whereas parenteral injections of large doses of liver extract regularly arrest it.

A deficiency of vitamin C causes an anemia which may become severe. This does not respond either to iron or liver preparations, but does to administration of the vitamin, which may stimulate a reticulocyte crisis, analogous to that seen after liver therapy in pernicious anemia.[11] There is no definite evidence that a lack of the other vitamins has a direct effect on blood formation, although the general ill health that results from such a deficiency may exert an indirect effect.

There is experimental evidence, however, that vitamin deficiency may affect gastric function and thus indirectly cause anemia. Cowgill[12] found that in dogs a deficiency in B_1 caused a subacidity or a complete achylia. Miller and Rhoads,[13] using a slight modification of the diet by means of which Goldberger produced pellagra and black tongue, induced in swine a deficiency disease resembling sprue and characterized by stomatitis, anemia (usually macrocytic in type), and achlorhydria. The intrinsic factor disappeared from the gastric juice and the anti-anemic principle from the liver.

It is evident from these facts that deficiencies of various kinds are the primary causes of many types of anemia. A poor diet may cause anemia because it lacks some of the raw materials from which the red cells are constructed. It may lack substances which, either directly or after further elaboration in the body, stimulate or control the processes of cell production. Finally it probably may lead to disturbance of function and possibly to degenerative changes in the gastro-intestinal tract which interfere with the absorption of these materials or prevent the elaboration of the anti-anemic principle.

A suitable diet is the first essential in the prevention of anemia. It must be adequate in protein, in vitamins and in mineral salts, particularly iron. This suffices in most cases, but if the function of the gastro-intestinal tract is disturbed, or if anemia has already developed, it is usually necessary to supplement the diet with iron or vitamins or liver, as the case may require. Of the various deficiencies discussed, the most important, because of its great frequency, is a lack of iron, particularly in infants and in women. It is easier to prevent such an anemia than to cure it, but it can usually be cured if the precise nature of the deficiency is recognized and if the necessary material is liberally supplied.

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X-RAY: FURTHER PROGRESS

From its discovery, the roentgen ray was seen to be valuable to physicians for disclosing the outline and density of the human skeleton, fractures and other bony defects. In the past forty years, as physicists have developed better x-ray machines, there would appear to be no other single agent upon which medicine has become more intimately dependent. The shape of hollow organs is now demonstrated by filling them with radiopaque solutions, such as the various iodine preparations. New growths, filling defects, and other anomalies of outline of thin tissue may thus be seen. Function is demonstrated as well as form, in various ingenious ways. The gallbladder which does not concentrate tetraiodophenolphthalein is considered diseased though it shows no stone. The kidney which does not excrete iopax has usually other functional defects. Foreign bodies in hollow organs were demonstrated first only if they were radiopaque. Later, methods were devised for demonstrating the radiolucent as well. Even the moving picture industry has affected roentgenology. X-ray movies have been made to show the internal organs in action, and the cinemas are more instructive than are single plates. A further development of the x-ray movie is the cinefluorograph, a moving picture of the fluoroscopic image, described by Stewart and Ghiselin[1] in this issue of the JOURNAL. Moving pictures are taken during fluoroscopy, which show digestive physiology and pathology, heart movements, and so on, and which may be studied intensively at one's leisure without subjecting patient or physician to prolonged radiation. The method has the advantage that the film is less costly than the x-ray film. As Hodges[2] says, the pictures demonstrate pathologic conditions in the esophagus, stomach, colon, lungs, and other parts of the body, in an almost hair-raising manner.

If one turns from the field of x-ray diagnosis to that of therapy, equally rapid advances show from year to year. The roentgen ray is an integral part of many dermatologic methods. Acne has been treated with considerable success for more than twenty years. Many other skin blemishes are benefited. X-ray therapy of malignancy is constantly expanding. The treatment of localized infections would seem also to be increasing in popularity. In this issue of the JOURNAL is a paper by the late Willis F. Manges,[3] formerly Professor of Roentgenology at Jefferson Medical College, Philadelphia, a pioneer who has contributed to the progress of radiologic technic from the beginning of this century. Manges claims benefit only for localized infections, as carbuncles, erysipelas, and osteomyelitis. He does not report any success in treating, for example, infectious diseases of childhood. The popularity a few years ago will be recalled of x-radiation for whooping cough. It was apparently short-lived. According to Manges, x-radiation is useful wherever heat or poultices were formerly employed.

1. Stewart, W. H.; and Ghiselin, F. H.: Recent Developments in the Cinematography of the Fluoroscopic Image. THE JOURNAL, this issue, p. 268.
2. Hodges, Fred M.: The Roentgen Ray: Facts and Fiction. THE JOURNAL, **29**:1151 (Dec.) 1936.
3. Manges, Willis F.: The Roentgen Treatment of Infections. THE JOURNAL, this issue, p. 244. Editorial: Amer. Jour. Roentgenology and Ra. Ther., **37**:106 (Jan.) 1937.

POSTERIOR PITUITARY EXTRACTS IN ECLAMPSIA

Water, of course, makes up two-thirds of the body weight and is its most abundant and essential constituent. Water metabolism has been extensively studied by surgeons because in surgical emergencies maintenance of fluid balance often means the difference between life and death. Fluids must be given to restore the large amounts lost during operative periods through bleeding, through the urine, breath, or vomiting. The obstetrical patient is generally advised to take plenty of fluids, but as a rule there is less careful quantitive study of fluid loss or retention, unless oliguria is notable. Extracts of the posterior pituitary gland are commonly employed at the time of delivery to produce uterine contractions, without great regard to whether this product alters the fluid and mineral exchange of the tissues. It is said to decrease the volume of urine and to increase the urinary chlorides, but to cause little or no rise in blood pressure in the normal non-pregnant individual.

According to Dieckmann and Michel,[1] of the University of Chicago, it has quite different effects in toxemia of pregnancy. It causes a rise in blood pressure of around fifty millimeters of mercury in eclamptics and pre-eclamptics. The pre-eclamptic patient, they say, reacts to posterior pituitary injections with oliguria, increased blood pressure, and occasionally cerebral symptoms and visual signs. Since it causes no rise of blood pressure in normal pregnancy, the susceptibility to posterior pituitary extracts may be used to differentiate the eclamptic state from primary hypertension. The above authors suggest that eclampsia and albuminuria of pregnancy are endocrine in origin, the posterior pituitary dominating the picture. This product therefore may be dangerous and should not be given to toxemic patients. The forcing of fluids in these cases also may be harmful.

1. Dieckmann, W. J.; and Michel, H. L.: Vascular Renal Effects of Posterior Pituitary Extracts in Pregnant Women. Amer. Jour. Obst. and Gyn., **33**:131-7 (Jan.) 1937.

A cold pressor test was described a few years ago, which is positive in primary vascular renal disease and possibly in patients with a tendency to the development of hypertension, or a hypertensive diathesis.[2] It is a rather simple test depending upon blood pressure determinations before and after brief immersion of the patient's hand in cold water. Patients with hypertension or the tendency to it respond with an unusual rise in blood pressure after this slight stimulus. Pre-eclamptics, according to the Chicago investigators, show only a moderate rise during the cold test, though they are very sensitive to posterior pituitary.

Characteristics of pre-eclampsia then, are the slight or moderate rise of blood pressure in response to the cold stimulus, and a marked rise after posterior pituitary injection. The essential hypertensive should be very susceptible to a slight cold stimulus, the pre-eclamptic to posterior pituitary products.

If confirmed, the studies are of much practical interest in obstetrics.

GLEANINGS FROM RECENT JOURNALS

Development of Tuberculosis in Adult Life.—In studies of the development of tuberculosis after childhood, Myers and his associates[3] in Minneapolis have examined the records of more than twelve thousand students over a period of years. A comparatively small proportion of these were medical students and nurses, but among this group occurred an overwhelming preponderance of the cases of tuberculosis. Many individuals reached college age with negative tuberculin reactions. The first evidence of developing disease was the appearance of a positive skin reaction in persons previously negative. At this time no evidence of tuberculosis appeared in the x-ray.

2. Hines, E. A.; and Brown, G. E.: A Standard Test for Measuring the Variability of Blood Pressure; Its Significance as an Index of the prehypertensive State. Ann. Int. Med., **7**:209, 1933. Ed., South. Med. Jour., **28**:388, 1935.

3. Myers, J. Arthur; Diehl, H. S.; Boynton, R. E.; and Trach, B.: Development of Tuberculosis in Adult Life. Arch Int. Med., **59**:1 (Jan.) 1937.

Physicians and nurses of course should be the strongest forces in prevention of the spread of any infection, particularly of this widespread disease. Every safeguard should be provided for those entering the healing profession. It would appear that the cases among medical students and nurses developed from exposure during routine work. One would immediately suspect that they were not taught a sufficiently aseptic technic in the handling of open cases.

The Minneapolis investigators advocate a tuberculin test every six months for those who may be in any way exposed to the infection. First infection of adults, they say, is in every way as benign as first infection in childhood.

Vitamins and Bleeding Tendency.—One of the main divisions of the vitamin classification is between the fat soluble and the water soluble. A, D, and E are fat soluble; C, G and the various B's are water soluble. Deficiency in water soluble C causes scurvy and in this disease there is abnormal bleeding. Last year a new vitamin was reported by Danish investigators,[1] a fat-soluble one, deficiency of which was accompanied by severe bleeding of chickens. For it the designation K was suggested. After slight injury, as by the removal of a pin feather, the K-deficient chicks often bled to death. The condition somewhat resembled human hemophilia. The vitamin was abundant in fresh cabbage, hempseed and hog liver. It was not found by them in cod liver oil, and thus was differentiated from vitamins A and D.

According to workers in Berlin,[2] another fat-soluble vitamin, for which the designation vitamin T was suggested, increased the platelet count in a child, or caused thrombocytosis in a case of thrombopenia. In a normal child also it increased the platelet count. Vitamin T occurred in several vitamin A containing foods, but not in cod liver oil or olive oil. Sesame oil was the source of a concentrated preparation.

Investigators will now have to continue the studies to prove that vitamin K is not vitamin T, since both are fat-soluble and both are said to influence the clotting of the blood. The last generally used vitamin designation is G. The committe on vitamin nomenclature will have to function to show cause why, in the naming of the anti-bleeding factor, even if K and T are separate and distinct, all intervening letters should be skipped.

Kidneys and Parathyroids.—In rickets, phosphorus and calcium metabolism are often abnormal, and rickets is practically a universal disease. The kidneys, of course, are responsible for excretion of large quantities of the two elements, and the parathyroid glands are intimately concerned in the regulation of their level in the blood. The parathyroids are enlarged in rickets. About half a considerable series of patients with parathyroid tumor were shown to have damaged kidneys; and conversely, the parathyroids of nephritics are reported to be fifty per cent larger than those of non-nephritics. Because of the frequency of rickets and of nephritis, with both of which abnormalities of parathyroid tissue may be associated at autopsy, one would expect parathyroid disease to be more common than clinical reports so far have indicated. There is every reason to believe that cases are escaping discovery.

Various methods are employed in the attempt to diagnose parathyroid disorder. One is by studies of the blood calcium and phosphorus. One is by testing the quantity of hormone in the circulating blood. A considerable quantity of blood is taken from the suspect patient, injected into a rabbit, and the effect in raising the blood calcium is noted. According to Highman and Hamilton,[1] of the University of Chicago, all but three of twenty-three nephritics so tested showed an abnormal increase of parathormone in the blood.

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1. Editorial, Experimental Hemophilia. South. Med. Jour., **29**: 1145 (Nov.) 1936.
   Almquist, H. J.: Purification of Antihemorrhagic Vitamin Jour. Biol. Chem., **114**:241, 1936.
2. Schiff, E.; and Hirschberger, C.: Thrombocytosis Produced by a Hitherto Unknown Substance, Fat Soluble T Factor. Am. Jour. Dis. Child., **53**:32 (Jan.) 1937.

1. Highman, W. J., Jr.; and Hamilton, Bengt: Hyperparathyroidism in Kidney Disease. Jour. Clin Invest., **16**:103 (Jan.) 1937.

Blood Sugar Rise After Encephalography.—An effect of piqure of the fourth ventricle of the brain of dogs is to cause onset of temporary diabetes, as Claude Bernard showed in 1849. It is of interest that a rise of blood sugar to as much as two hundred per cent of the former level is frequent in human beings after encephalography.[1]

During this diagnostic process, of course, the spinal fluid is removed and replaced with an equal volume of air, following which pictures are taken. The rise of blood sugar is apparently constant, occurs fifteen to twenty minutes after the cavities are filled with air, and the return to normal is much slower. The rise would suggest some variety of stimulation, possibly trauma, to the ventricle.

TWENTY-FIVE YEARS AGO
From Journals oe 1912

"Alcoholism among the Jews is an almost unknown affection.[2] Dr. Norman Kerr (Inebriety: Its Etiology, etc.) says of the Jews: 'Extensive as my professional intercourse with them has been, I have never been consulted for inebriety in the person of a Jew, while my advice has been sought for this complaint by a very large number of Christians . . . Of the strong impulse to alcoholism or other narcotism, I have never seen a case amongst this distinctive people.'"

Endorsement of the Council on Chemistry and Pharmacy.[3]—"Reflections are inspired by expressions in certain medical journals which betray an unfriendly feeling towards the A.M.A. Council on Chemistry and Pharmacy, and to the Journal of the Association.

"Frequently sneering allusions are indulged in at the expense of the fallibility of the judgments published by the Council.

"Of course we are not so irreverent as to compare those judgments with the rules and guide for our conduct laid down in the Bible as any way similar in authority. We simply desire to emphasize the truth that *each in its own way is the highest available authority.* If we refuse to be guided by them only confusion and anarchy could prevail in their relative realms."

1. Bradley, Charles: The Effect of Encephalography on Blood Sugar Level of Children. Amer. Jour. Med. Sci., **193**:259 (Feb.) 1937.
2. Wolff, Bernard: Are the Jews Immune to Pellagra? South. Med. Jour., **5**:121, 1912.
3. Editorial: Reasons Why Doctors Should Endorse the Findings of the A.M.A. Council. South. Med. Jour., **5**:278, 1912.

Correspondence

AN EFFICIENT AND INEXPENSIVE SKIN CLEANSING FORMULA*

Editor, Southern Medical Journal:

Patients have requested information at times concerning prescriptions which may be written by the physician to combat the high cost and exploitation of proprietary cosmetic preparations. It is our feeling that general practitioners of medicine should have simple formulae of the above nature available for their use. However, this information is not taught usually in medical schools.

Patients are usually grateful for advice on these matters. They have had to pay an excessive amount of money for a good skin cleansing agent because some soaps are excessively drying, especially in cold weather.

In standard textbooks of dermatology, we were unable to find a satisfactory formula for a simple, inexpensive, and non-sticky cleansing solution. Therefore, we began experimentation to evolve such a formula.

We are publishing the result with the hope that the general practitioner will find it useful in the guidance of his female patients.

℞	Gm. vel C.C.
Oil of almond	0.05
Oil of lavender	0.75
Glycerine	2.00
Tincture of green soap	2.00
Tincture of cudbear	0.07
Alcohol (95 per cent ethyl) Q.S. ad	120.00

Mix thoroughly and write:

Apply to cotton pledget and clean face thoroughly morning and night.

Wallace Marshall, M.D.

University, Alabama, Dec. 3, 1936.

*From the Departments of Medicine and Physiological Chemistry, University of Alabama School of Medicine, University, Alabama.

Book Reviews

A Century of Medicine in San Antonio: The Story of Medicine in Bexar County, Texas. By Pat Ireland Nixon, M.D., San Antonio. Lancaster, Pennsylvania: Lancaster Press, Inc., 1936. Book may be purchased from the Bexar County Medical Society, 202 West French Place, San Antonio, Texas. Cloth $4.00; De Luxe edition $10.00 (100 copies only).

Fifty-three years ago the author, Dr. Patrick Ireland Nixon, was born in Nixon, Texas, and he has lived in Texas all his life except during the years that he spent in academic and medical colleges and in hospital training. His devotion to San Antonio, Bexar County and the Lone Star State is as ardent as was his Irish ancestors' affection for "the shamrock and the rose"

that grow on an island that is "a bit of heaven in this divil of a world." Dr. Nixon is a highly cultivated, lovable gentleman and an accomplished surgeon, with a penchant for history and literature. Therefore, by birth, education, training and temperament he is eminently fitted to write the medical saga of San Antonio. In charm of expression and in thrilling recital of the deeds of valor of "physicians who lived and wrought when Texas history was in the making," "A Century of Medicine in San Antonio" deserves to rank with historic and legendary stories of Icelandic heroes, and with the tales of chivalrous adventure that have come down to us from the Middle Ages. The author delved deep into the medical history of San Antonio from the time when it was an Indian village until it was immortalized in history by the martyrs of the Alamo. His delineation of the incantations and impositions of the Indian "medicine man," and the only slightly improved methods of treating the sick by Spanish surgeons who first came to San Antonio, seem to be based upon more than mere tradition.

The description of the part played by Dr. Amos Pollard and his two associates, Dr. D. Michison and Dr. John W. Thompson, who sacrificed their lives in the performance of duty with Travis, Bowie, Crockett and Bonham, in the Alamo makes a thrilling narrative. Of the three physicians who were butchered by Santa Anna's Mexican hordes, Dr. Nixon said: "History does not record the deeds of these medical martyrs on those memorable days in March, 1836, nor does it exalt their heroism, but it is not difficult to visualize them as they went quietly and unostentatiously about their work with full knowledge that eventual destruction of all was inevitable, giving solace and relief from pain to the end."

The chapter "When Texas Was a Republic," is excellent history and full of humorous incidents of the primitive practices in that era. Medicine in San Antonio from 1845 to the early fifties, in "the days of tallow candles and spinning wheels, ox teams and water mills," showed an upward trend. A few excellent physicians located in San Antonio in the forties. The Bexar County Medical Society was organized in 1853, and in 1861 San Antonio was ready to send a number of well prepared surgeons for service in the Confederate Army. Likewise, after the war the physicians of Bexar County, "reconstructed but unregenerate," did their part in the development of the great Southwest.

Beginning in the late eighties, San Antonio, having "a mild bracing climate in the health belt where consumption and catarrh are cured without medicine," attracted many sufferers from chronic lung diseases; and likewise there was an influx of highly trained physicians, some of whom came for their health, and having recovered, joined with others who came to practice medicine. The medical profession of Bexar County made remarkable progress in the nineties; and the dawn of the twentieth century found San Antonio physicians with the vanguard in the reorganization and the upbuilding of the Texas State Medical Association, which today is perhaps the greatest state medical organization in the entire United States. One of the Nestors of the medical profession of the Southwest was Dr. Frank Paschal of San Antonio, who was largely responsible for the establishment of a great modern state tuberculosis hospital at Carlsbad, Texas.

A delightful feature of "A Century of Medicine in San Antonio" is a collection of biographical sketches of "revered physicians of the twentieth century." Paschal, Hicks, Caffery, George H. Moody, Jackson, the Herffs, Shropshire ("Old Shrop"), McCamish ("the Will Rogers of Bexar County"), Dabney Berry and other beloved physicians still live in the hearts of their confreres. The San Antonio "roll of honor" among World War surgeons is impressive; and in the "five years of plenty" Bexar County doctors "risked all their winnings and lost," while in the "six lean years" that followed they "never said a word about their loss," but acquired a new home for their revered County Medical Society.

In the final chapter, "Yesterday, Today and Tomorrow," the author points with pride to the past and to the present, and he pays a fine tribute to the country doctor; and couched in typical Pat Nixonian artistic language, the closing paragraph should appeal to physicians everywhere:

"And to a sympathetic understanding we would ask the addition of a tolerant spirit. We of this generation humble ourselves before the life and work of our medical forefathers. When another century of medicine shall have rolled around and some historian records our medical deeds and misdeeds, we trust it is not too much to hope that our life and our work likewise will be exalted."

"A Century of Medicine in San Antonio" will provide profitable and relaxing entertainment for a number of evenings to any physician who enjoys reading good literature. It abounds in humor and is an excellent history of the Southwest for a hundred years. It is a beautiful volume, a credit to the book makers' art, that will delight medical bibliophiles from Maine to California. Members of the Southern Medical Association who enjoyed the charm of the old and new San Antonio and who were overwhelmed with the hospitality of Bexar County physicians during the 1934 meeting will read with added interest Dr. Nixon's delightful story of medicine in the Southwest.

Bright's Disease and Arterial Hypertension. By Willard J. Stone, B.Sc., M.D., F.A.C.P., Clinical Professor of Medicine, University of Southern California, Los Angeles; Attending Physician to the Pasadena Hospital, Pasadena, California. 352 pages, illustrated. Philadelphia: W. B. Saunders Company, 1936. Cloth $5.00 net.

This book opens with a short chapter summarizing some of the more important contributions to the subject of Bright's disease made in the past seven hundred years. The second chapter deals with the classification of this group of disorders, and the third with renal physiology. Then follows a series of chapters which are concerned with the general aspects of chemical and pathological changes produced in the body by diseases of the kidneys, and there are concise descriptions of the several stages of glomerular nephritis, of nephrosis and of primary hypertensive disease with and without renal complications. The book is well written, is up to date, has a clear style, and presents in a simple form a subject which is confusing to the minds of many physicians. Although it contains little that is original it can be highly recommended to those who are desirous of obtaining more information than can be found in the usual textbooks and who are prevented by limitations of time from reading the more comprehensive works dealing with the same subject.

Physical Diagnosis. By Ralph H. Major, M.D., Professor of Medicine in the University of Kansas, Kansas City. 457 pages, illustrated. Philadelphia: W. B. Saunders Company, 1937. Cloth $5.00.

Here is a book that is refreshingly new both in text and presentation. Dr. Major has omitted laboratory procedures, limiting the text to those findings which may be elicited by inspection, palpation, percussion and inspection. Not being satisfied with later day descriptions he has included a number of quotations from the writings of such men as Auenbrugger, Laennec, Skoda and others. There is a tendency throughout the book to be rather brief. This is especially noticeable in the description of abdominal findings, rectal examination and the lack of a description of bimanual examination in the female. The chapters on the heart and chest are especially pleasing. An unusually large number of good illustrations are exhibited. The writer impresses the student with the importance of physical diagnosis and gives him the means to gain a good basic training in common diagnostic procedures. The more skilled practitioner and internist requires a book of greater details.

Rural Health Practice. By Harry S. Mustard, M.D. Associate Professor, Public Health Administration, School of Hygiene and Public Health; Lecturer, Preventive Medicine and Public Health, School of Medicine, The Johns Hopkins University. 603 pages. New York: The Commonwealth Fund, 1936. Cloth $4.00.

Everything that the rural county health officer needs to know appears to be in this book, from a historical outline of the founding of the first county health units to the final chapter on "School Sanitation and Hygiene." There is information on how to organize the office, how to organize health projects, selection of quarters, relationship to the state health department program, finances, statistics, school examinations, nursing visits, isolation, diagnostic clinics and excreta disposal.

Dr. Mustard has had over twenty years of fruitful experience as this book shows. The new health officer using this book will find his work much easier and he will avoid many of the unpleasant situations that otherwise might occur. The more experienced practitioner will find many helps in the planning of his own programs. At the end of each chapter is a bibliography for further reading. An exceedingly helpful volume for anyone engaged in public health work.

Modern Treatment and Formulary. By Edward A. Mullen, P.D., M.D., F.A.C.S., Assistant Professor Pharmacology and Physiology, Philadelphia College of Pharmacy and Science, Lieutenant Commander, Medical Corps, U. S. Naval Reserve. Foreword by Horatio C. Wood, Jr., Professor of Therapeutics in University of Pennsylvania, Graduate School of Medicine, Professor of Pharmacology and Physiology, Philadelphia College of Pharmacy and Science. 707 pages. Philadelphia: F. A. Davis Company, Publishers, 1936.

This book contains the treatments for some of the rarer disease conditions and symptoms as well as those which are more common. These conditions are alphabetically arranged and under each heading is a description of the accepted treatment and a large number of prescriptions. These prescriptions are varied, being both old and recent. Many are signed. This section is also cross indexed as several pathological states may exhibit the same symptoms.

The appendix contains notes on intravenous and hypodermic medication, diet lists, liquid food formulae, treatment of fractures, sprains and dislocations, hints on prescribing, chart of eruptive fevers, a differential diagnosis table, dose table, weights and measures, information on the female pelvis and uterus, conception periods, hemorrhages and wounds, miscellaneous emergencies and a five-language physician's interpreter. An authoritative and useful work.

Operative Surgery. By Alexander Miles, M.D., LL.D., F.R.C.S., Ed. Consulting Surgeon, Royal Infirmary Edinburgh, and D. P. D. Wilkie, M.D., F.R.C.S., Ed. and Eng. Professor of Surgery, University of Edinburgh. Second edition. 631 pages, illustrated. London, England: Oxford University Press, 1936.

From the preface of the first edition of this work, the following paragraphs are quoted:

"In this work, which replaces the companion volume of our 'Manual of Surgery,' we have not attempted to cover all the ground, but have been content to provide for the needs of undergraduates who require a guide to their studies in the class of operative surgery and in the hospital, and of young graduates who may be called upon to undertake operative work as house surgeons or in practice.

"The methods selected for description are those most generally favored by the contributors. Limitation of space alone has prevented alternative methods being described in detail. The text may therefore be taken to represent in a general way the present-day practice in the Edinburgh School."

These quotations indicate the character and limitations of the volume. For the purposes above outlined, the work is invaluable. It describes lucidly methods of attacking the manifold surgical problems which are daily encountered in every large hospital.

The reviewer feels that he may go further than the authors, who specially recommend the work to "young graduates and house surgeons," and state that it will prove a valuable guide to all surgeons as a ready reference work on operative surgery.

Physical Therapeutic Methods in Otolaryngology. By Abraham R. Hollender, M.D., F.A.C.S., Associate in Laryngology, Rhinology, and Otology, University of Illinois College of Medicine; Fellow of the American Academy of Ophthalmology and Otolaryngology. 442 pages, illustrated. St. Louis: The C. V. Mosby Company, 1937. Cloth $5.00.

This work deserves the hearty approval of the otolaryngologist for several reasons. The author has first surrounded himself with a group of excellent collaborators. The chapter dealing with fundamentals of physiotherapy is excellently presented and should very definitely clarify the haze that has clouded the minds of the "unbelievers" regarding this form of medical practice. Technics are clearly described and there are many excellent illustrations.

Current literature for the past ten years is carefully reviewed. The work as a reference deserves a place in the library of all otolaryngologists.

A Textbook of Medicine. By Charles Phillips Emerson, M.D., Research Professor of Medicine, Indiana University, Formerly Associate in Medicine and Medical Resident, Johns Hopkins University and Hospital; Assistant Professor of Medicine, Cornell University (Ithaca); Medical Superintendent, Clifton Springs Sanitarium, New York; Professor of Medicine and Dean, Indiana University School of Medicine, Indianapolis, Indiana. 1296 pages. Philadelphia: J. B. Lippincott Company, 1936. Cloth $8.00.

This book is necessarily large since it covers practically the whole field of medicine in an exceptionally thorough way.

It differs from many books in which some diseases are considered exhaustively while others are barely touched, in that each disease receives the space necessary for concise coverage.

The general grouping of diseases follows the usual plan of textbooks of medicine: "Specific Infectious Diseases," "Diseases Caused by Animal Parasites," "Diseases Due to Drugs and Poisons," "Diseases of Metabolism," etc., through twenty major divisions to "Psychoneurotic Disorders." Under each major division the individual disease or conditions of that group are considered, and a uniform plan of presentation is followed for all. This embraces: definition, incidence, symptoms, diagnosis, prognosis and treatment. In addition to these a short "Historical Note" is frequently added, and, when pertinent, the "Morbid Anatomy." To each disease or condition considered has been added a convenient list of references. Prophylaxis and community problems are considered briefly.

The book is to be commended as a text and reference, particularly for symptoms and treatment.

Tissue Immunity. By Reuben L. Kahn, M.S., D.Sc., University of Michigan, Ann Arbor. 707 pages, illustrated. Springfield, Illinois: Charles C. Thomas, 1936. Cloth $7.50.

This book, a comprehensive treatise on immunology, contains experimental data which have been introduced to illustrate the various points under discussion. Following an introductory chapter the author devotes seventeen chapters to: Tissues in the non-immune state, in the period of incubation, in the immune state, as immunity increases, as immunity subsides, in the disimmune state, in natural and passive immunity, in the young; immunological differences of different tissues; phenomena of tissue necrosis; specific tissue reactions with antigens; specific tissue reaction and antibodies; specific tissue reaction and non-specific factors; immunologic effect of the specific tissue reaction; theoretical and practical aspects of tissue immunity.

The chapters are admirably arranged. Each is briefly outlined, contains an introduction, and ends with a summary and a discussion of clinical considerations. Explanations are, for the most part, clear and logical.

The author believes that allergic manifestations are expressions not of specific hypersensitiveness but of specific hyperimmunity, and that the outstanding disturbance in the allergic person is an immunologic response to substances that are not antigenic to normal persons. He offers explanations of how serums are effective when given intravenously, why children can withstand more biologic therapy than adults, why children are more susceptible to certain diseases and less to others. He offers evidence to show that small doses of antigen are preferable to large doses, and that vaccine given in too frequent doses will produce specific immunological fatigue.

In a comprehensive study of immunology in which many controversial subjects are discussed it is not surprising that conclusions are occasionally at discord with others. The book is a valuable contribution to a fundamental medical science and will unquestionably do much to stimulate investigation.

International Health Division Annual Report, 1935. 286 pages, illustrated. New York: The Rockefeller Foundation.

This report of the activities of the International Health Division is considerably more detailed than that covered in the general report of the Rockefeller Foundation for 1935, and is intended for distribution to those having special interest in public health.

It is a useful reference, and a stimulating account of progress in public health. Most of the report is devoted to a very instructive summation of field and laboratory research on yellow fever, malaria, hookworm disease, tuberculosis and yaws. There is presented also a digest of the research work performed by the Division, or under its auspices. References are cited to 111 papers published in the medical press.

The International Health Division has continued its program of aiding various countries and states through the installation and demonstration of improved public health methods and organization. There has been real assistance in furthering the development of schools of public health, in the establishment of training centers, and in providing an extensive program of fellowship for advanced workers in public health.

A table outlines various expenditures which totaled $1,916,303 for the year.

Collected Writings. By Alfred F. Hess. Volumes I and II. 719 pages, illustrated. Springfield, Illinois: Charles C. Thomas, 1936. Cloth $15.00.

The fame of the late Alfred F. Hess as an investigator and contributor to pediatrics is well known to his colleagues and the publication of the greater part of his contributions by Mrs. Hess will serve to keep green the memory of his profound influence on its development, not only in this country but in the world. A glance at the table of contents will show that Dr. Hess investigated many problems in pediatrics at different periods of his busy life and always followed up the particular subject under consideration until its possibilities were exhausted. To appreciate his enormous energy and intellectual honesty it is necessary to read his papers in detail. Dr. Hess is best known for his researches in scurvy. He showed that vitamin C was destroyed by oxidation, especially in an alkaline medium. He also demonstrated the destructive action of heat and aging on the vitamin. The discovery of the important factor of oxidation led to the modern method of canning and preserving of various foods in which the vitamin is preserved. Of equal importance are his investigations on rickets. His carefully controlled experiments demonstrated beyond doubt the efficacy of cod liver oil as a rich source of vitamin D in the pre-

vention and cure of this disease. His discovery of the synthesis of vitamin D in food substances through irradiation with ultraviolet light ranks as one of the greatest discoveries in medicine. He also demonstrated the fact that the vitamin D content of milk could be increased by feeding of yeast to dairy herds. From this has grown the development of vitamin D milk, which is available commercially. In addition to his contributions to vitamin researches, Dr. Hess was tremendously interested in the problem of tuberculosis in infancy. His papers on this subject resulted in the establishment of the first sanatorium for infants already infected or living in an environment which subjected them to almost certain infection. After a perusal of these collected writings one cannot help but have a feeling of profound regret that the work of this great mind has been cut short in its prime. And Mrs. Hess should be commended for the publication of these volumes which commemorate the life work of her late husband.

An Index of Differential Diagnosis of Main Symptoms. By various writers; edited by Herbert French, C.V.O., C.B.E., M.A., M.D., Oxon., F.R.C.P. (Lond.), Consulting Physician to Guy's Hospital; late Physician to H. M. Household. Fifth Edition. 1,145 pages, illustrated. Baltimore: William Wood & Company, 1936. Cloth $16.00.

This book preserves the characteristics that have proven desirable in previous editions, that is, the grouping of symptoms together and a leaning toward clinical rather than laboratory knowledge. While the information given is not complete under any one heading, the reader may acquire a rather comprehensive knowledge of a disease by referring to the index and reading in various portions of the book. A number of the newer diagnostic tests are included and some of the older ones discarded. An unusually large number of illustrations and color drawings give the reader a vivid picture of the pathology involved.

Security Against Sickness: A Study of Health Insurance. By I. S. Falk. 423 pages. Garden City, New York: Doubleday, Doran & Company, Inc., 1936. Cloth $4.00.

I. S. Falk, Ph.D., the writer of this well documented book, was a member of the Committee of the Cost of Medical Care and health consultant to the President's Committee on Economic Security. Health insurance is defined as, "the pooling of funds for a group of persons so that each is assured protection against the insured risk (loss of wages and cost of medical care or both) under a government - supervised, non - profit scheme." The cost of sickness, of preventable deaths, of loss of earnings and medical costs are discussed, stating that though the nation should save 20 per cent in its drug bill we should still have to find solutions for, "(1) The uncertain, uneven and unbudgetable size of medical costs for individuals or families; (2) the difficulty of knowing how, when and where to secure good medical care; and (3) the uncertain and inadequate remuneration of practitioners and institutions."

The second part is a study of the European experience with health insurance and specifically described are the forms found in Germany, Great Britain, France and Denmark. From the trend of arguments concerning each system one would gather that Dr. Falk is not only an ardent partisan for health insurance but that he has little time for the "petty criticisms" of his opponents.

The third part offers a basis for an American plan of health insurance. Five basic principles are given: "(1) So far as may be practical, economic security should be assured against the costs of medical care and against the loss of earnings caused by disability; (2) the provision of good medical care to all the population is essential to the nation's well-being; (3) the costs of sickness (medical care and loss of earnings) should be distributed among groups of people and over periods of time; (4) those who render medical care should be adequately remunerated, and (5) quality in medical care should not be sacrificed to economy in cost."

There are four additional principles as follows: "(6) Regulations and supervision of the qualification of practitioners and institutions are public functions; (7) the provision for medical care for the dependent or indigent sick is an obligation of society; (8) the medical care of non-indigent persons afflicted with serious mental disorders or with tuberculosis or with any other disease dangerous to public health (such as syphilis or gonorrhea), is a concern of society and falls within the domain of public health practice, and (9) the medical care of non-indigent persons afflicted with other diseases (for example cancer and orthopedic conditions) has tended increasingly to become a public responsibility when elaborate or expensive facilities are required for their diagnosis and treatment."

In the fourth portion are a number of different principles and resolutions adopted by such organizations as the American Medical Association, American College of Surgeons, American Hospital Association and others.

There is no doubt that this book is a definite contribution to the study of health insurance. There is no doubt that Dr. Falk proposes a very convincing set of arguments for health insurance. There is no doubt that the benefits to be derived from health insurance are presented in a partisan manner. But there is doubt on the part of the medical profession and a great many others that it is the Utopia.

Dr. Falk expects to cut the costs without cutting the quality of medical service. He says that a panel of 2,500 will guarantee the practitioner an income of $5,474, but ten lines above this is the statement that the average practitioner actually received in 1934, $2,065. Is this adequate remuneration? And how can a physician give adequate medical care to 1,000 people and to their dependents, about 4,000 people. It is small wonder that preventive medicine has not made much headway under health insurance. There is about one physician for every 800 persons in this country. In Great Britain many persons are referred to hospitals for specialist treatments, and when the patient cannot pay these men receive no remuneration. In Germany there has been a large influx of quacks and in France the *caisses* themselves are employing doctors to check up on the practitioners. If health insurance is satisfactory why do these practices occur and if it is so good why must it be made compulsory and not stand on its own merits?

This volume is not the answer to the question but a means of argument and should be read by everyone interested in health insurance. But it must be remembered that there are other views, and each is worthy of being heard and considered.

Southern Medical News

SOUTHEASTERN SURGICAL CONGRESS

The Eighth Annual Assembly of the Southeastern Surgical Congress, which was originally announced would be held in Louisville, Kentucky, on account of the flood situation has been changed to Charlotte, North Carolina, and will be held on March 8-10 at the Hotel Charlotte. The program as planned for Louisville will be carried out at Charlotte. There will be three full days of intensive postgraduate lectures and one public meeting with thirty-seven of the country's most outstanding surgeons making presentations. Each day at noon there wil be round table luncheon meetings and the regular annual banquet will be on Tuesday evening. For program and other information write Dr. B. T. Beasley, 701 Hurt Building, Atlanta, Georgia.

THE NEW ORLEANS GRADUATE MEDICAL ASSEMBLY

The New Orleans Graduate Medical Assembly will hold its First Annual Meeting in New Orleans at the Roosevelt Hotel, March 8-11. There will be clinical sessions with presentations by outstanding clinicians and teachers from the leading medical centers of the country. Luncheons and round table discussions will be held each day with the program on Monday evening in conjunction with the Orleans Parish Medical Society. All sessions and technical and scientific exhibits will be held at the Roosevelt Hotel. Dr. Hyder F. Brewster is General Chairman and officers of the Assembly are: Dr. Thomas Benton Sellers, President; Dr. Lucien A. LeDoux, Vice-President; Dr. William H. Gillentine, Secretary; Dr. Francis E. LeJeune, Treasurer; Dr. Frederick L. Fenno, Director of Clinics.

DALLAS SOUTHERN CLINICAL SOCIETY

The ninth annual Spring Clinical Conference of the Dallas Southern Clinical Society will be held at Dallas, Texas, March 15-18, at the Adolphus Hotel, conference headquarters. General assemblies, afternoon clinics, round table luncheons, clinical-pathological conferences, evening symposia, postgraduate lectures will be held with a smoker on Monday evening and a clinical dinner with entertainment on Thursday evening.

ALABAMA

Dr. Harry Katz, Birmingham, has been made Acting Health Officer of Macon County.

Dr. Eldridge T. Norman, Linden, and Mrs. Janie T. Harrison were married December 1.

Deaths

Dr. John Ernest Drakeford, Tuskegee, aged 65, died October 12.

Dr. Frank Paul Pettey, Decatur, aged 69, died November 26.

Dr. Joseph M. Steiner, Greenville, aged 57, died recently from injuries received in a fall.

Dr. E. B. Bailey, Demopolis, aged 61, died January 5.

Dr. Samuel L. Edwards, Tuscaloosa, aged 66, died December 30.

ARKANSAS

The Carroll County Medical Society has elected the following officers for the coming year: Dr. C. W. Slusser, Green Forest, President; Dr. J. F. John, Eureka Springs, Vice-President; Dr. D. K. McCurry, Green Forest, Secretary-Treasurer.

The Crawford County Medical Society has elected the following officers for the coming year: Dr. J. R. Crigler, Alma, President; Dr. M. S. Dibrell, Van Buren, Vice-President; Dr. O. J. Kirksey, Mulberry, Secretary-Treasurer.

The Franklin County Medical Society has elected the following officers for the coming year: Dr. W. C. Porter, Ozark, President; Dr. W. H. Gibbons, Ozark, Vice-President; Dr. Thomas Douglas, Ozark, Secretary-Treasurer.

The Independence County Medical Society has elected the following officers for the coming year: Dr. Paul H. Jeffery, Bethesda, President; Dr. L. T. Evans, Batesville, Vice-President; Dr. C. A. Churchill, Batesville, Secretary-Treasurer.

The Miller County Medical Society has elected the following officers for the coming year: Dr. R. R. Dale, Texarkana, President; Dr. W. D. Smith, Texarkana, Vice-President; Dr. R. R. Robins, Texarkana, Secretary-Treasurer.

The Nevada County Medical Society has elected the following officers for the coming year: Dr. A. B. Dickey, Prescott, President; Dr. S. J. Hesterly, Prescott, Vice-President; Dr. F. W. Regnier, Little Rock, Secretary-Treasurer.

The Saline County Medical Society has elected the following officers for the coming year: Dr. M. M. Blakely, Benton, President; Dr. J. W. Ashby, Little Rock, Vice-President; Dr. T. C. Watson, Benton, Secretary-Treasurer.

The Union County Medical Society has elected the following officers for the coming year: Dr. Berry L. Moore, El Dorado, President; Dr. W. S. Riley, El Dorado, Vice-President; Dr. J. K. Sheppard, El Dorado, Secretary-Treasurer.

The White County Medical Society has elected the following officers for the coming year: Dr. M. C. Hawkins, Jr., Searcy, President; Dr. C. M. Peeler, Pangburn, Vice-President; Dr. A. J. Dunklin, Searcy, Secretary-Treasurer.

Dr. John M. Smith, Russellville, has been assigned to the Conway County Health Unit.

Dr. D. K. McCurry, Green Forest, has been elected President of the First National Bank of that city.

Dr. W. A. Purifoy has been elected President of the Bank of Chidester.

Dr. C. J. Higinbotham, Pine Bluff, has been elected Surgeon of the Walter C. Hudson Camp, Spanish War Veterans.

Dr. John R. May, Little Rock, has been made Superintendent of the Pulaski County Hospital.

Dr. Thomas C. Watson, Benton, has been made Director of the Tenth Health District.

Dr. J. N. Roberts, Little Rock, has been appointed Deputy Coroner of Pulaski County.

Dr. Walter W. Brown, Williford, has been made Coroner of Sharp County.

Dr. George R. Siegel, Clarksville, has been made Coroner of Johnson County.

Dr. Bert B. Bruce, Alma, has been made Coroner of Crawford County.

Dr. J. T. Matthews, Heber Springs, has been made President of the Rotary Club of that city.

Dr. F. A. Corn, Jr., Lonoke, has been made a Trustee of the University of Arkansas.

Dr. J. A. Summers, Little Rock, has been made Director of the Pulaski County Health Unit.

Dr. W. Meyers Smith, Morrilton, has been made Director of Maternal and Child Welfare Division of the State Board of Health.

Dr. J. O. Pierce, Marked Tree, has returned after taking postgraduate work in Chicago.

Dr. Robert H. Johnson announces his association with Dr. Earle H. Hunt, Clarksville.

Dr. T. J. Stewart, Wynne, and Miss Myrtle Tyer were married December 23.

Deaths

Dr. Wylie E. Oldham, Calico Rock, aged 76, died November 19.

Dr. Henry Oscar Marcum, St. Charles, aged 67, died recently.

Dr. Joseph Griffin Waldrop, Hot Springs National Park, aged 63, died January 17.

Dr. Davis Ewing Evans, Harrison, aged 81, died January 10.

Dr. John Albert Burnett, Waldron, aged 68, died recently of uremia.

Dr. John Rainey Parker, Berryville, aged 59, died January 12.

Dr. James Knox Smith, Texarkana, aged 58, died December 28 from a fracture of the cervical vertebrae.

DISTRICT OF COLUMBIA

Rear Admiral Charles S. Butler, Medical Corps, United States Navy, delivered the Smith-Reed Russell Lecture at the George Washington University School of Medicine on January 21.

Deaths

Dr. Arthur Patterson Chadbourne, Washington, aged 73, died recently.

Dr. Adelbert A. Taylor, Washington, aged 67, died November 1 of chronic myocarditis.

Dr. Francis Edward Park, Washington, aged 82, died November 12 of injuries received in a fall.

Dr. Frank Austin Swartwout, Washington, aged 69, died December 16 of heart disease.

Dr. John H. Junghans, Washington, aged 68, died recently of cerebral embolism and arteriosclerosis.

FLORIDA

The Dade County Medical Society has elected the following officers for the coming year: Dr. R. N. Burch, Miami, President;

Continued on page 32

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Continued from page 354

Dr. John D. Milton, Miami, Vice-President; Dr. Walter C. Jones, Jr., Miami, Secretary; Dr. Sheffel Wright, Miami, Treasurer.

The DeSoto-Hardee-Highlands Medical Society has elected the following officers for the coming year: Dr. G. H. McSwain, Arcadia, President; Dr. W. H. Peacock, Wauchula, Vice-President; Dr. L. W. Martin, Sebring, Secretary-Treasurer.

The Escambia County Medical Society has elected the following officers for the coming year: Dr. J. C. McSween, Pensacola, President; Dr. Rufus Thames, Milton, Vice-President; Dr. J. M. Hoffman, Pensacola, Secretary-Treasurer.

The Orange County Medical Society has elected the following officers for the coming year: Dr. H. F. Harms, Orlando, President; Dr. H. A. Day, Orlando, Vice-President; Dr. Hewitt Johnston, Orlando, Secretary; Dr. Spencer A. Folsom, Orlando, Treasurer.

The Victoria Hospital Staff, Miami, has elected the following officers for the coming year: Dr. W. A. Haggard, President; Dr. Taylor Lewis, Vice-President; Dr. James J. Nugent, Secretary.

Dr. W. M. Rowlett, Tampa, has been appointed a Colonel on the Governor's Staff.

Dr. Reddin Britt, St. Augustine, has been elected President of the St. Johns County Welfare Federation.

Dr. Orville N. Nelson, Bay Pines, has been made a Fellow in the American College of Surgeons.

Dr. Henry L. Tippins, formerly of Savannah, Georgia, and Jacksonville, Florida, has returned from doing postgraduate work in Chicago and has opened offices in Miami, for the practice of pediatrics.

Dr. J. M. Gorman, Jacksonville, has moved into the Peninsular Life Building.

Dr. W. L. Fitzgerald, Miami, has moved into the Ingraham Building.

Dr. Henry B. Oertel, formerly of Willow, has moved to Osceola.

Dr. Ralph W. Jack, formerly of Farmville, Virginia, has moved to Miami.

Dr. A. G. Holmes, formerly of Daytona Beach, has moved to Miami.

Dr. B. B. Sory, Jr., formerly of West Palm Beach, has moved to Palm Beach.

Dr. F. A. Gowdy, formerly of Fort Pierce, has moved to Miami Beach.

Dr. W. O. Arnold and Miss Olive Edith Schell, both of West Palm Beach, were married December 5.

Dr. John O. Barfield and Miss Edith Herlong, both of Miami, were married December 1.

Deaths

Dr. Andrew Leight Monroe, River Junction, aged 80, died November 17 of hypertensive heart disease.

Dr. Roy Hayman McKay, Miami, aged 53, died November 28 of injuries received in a fall.

Dr. Charles Robert Sneath, Lakeland, aged 79, died November 14 of coronary thrombosis.

Dr. John Louis Seebold, LaBelle, aged 65, died November 14 of acute dilatation of the heart.

Dr. M. S. G. Abbott, Pensacola, aged 81, died November 7.

Dr. Genous Sanders Hodges, Marianna, aged 54, died December 9 of pneumonia.

Dr. Frederick Dracass, St. Petersburg, aged 89, died December 3 of arteriosclerosis.

GEORGIA

The Bartow County Medical Society has elected the following officers for the coming year: Dr. W. E. Wofford, Cartersville, President; Dr. J. W. Stanford, Cartersville, Vice-President; Dr. T. Lowry, Cartersville, Secretary-Treasurer.

The Carroll County Medical Society has elected the following officers for the coming year: Dr. H. J. Goodwyn, Carrollton, President; Dr. C. L. Baskin, Bremen, Vice-President; Dr. D. S. Reese, Carrollton, Secretary-Treasurer.

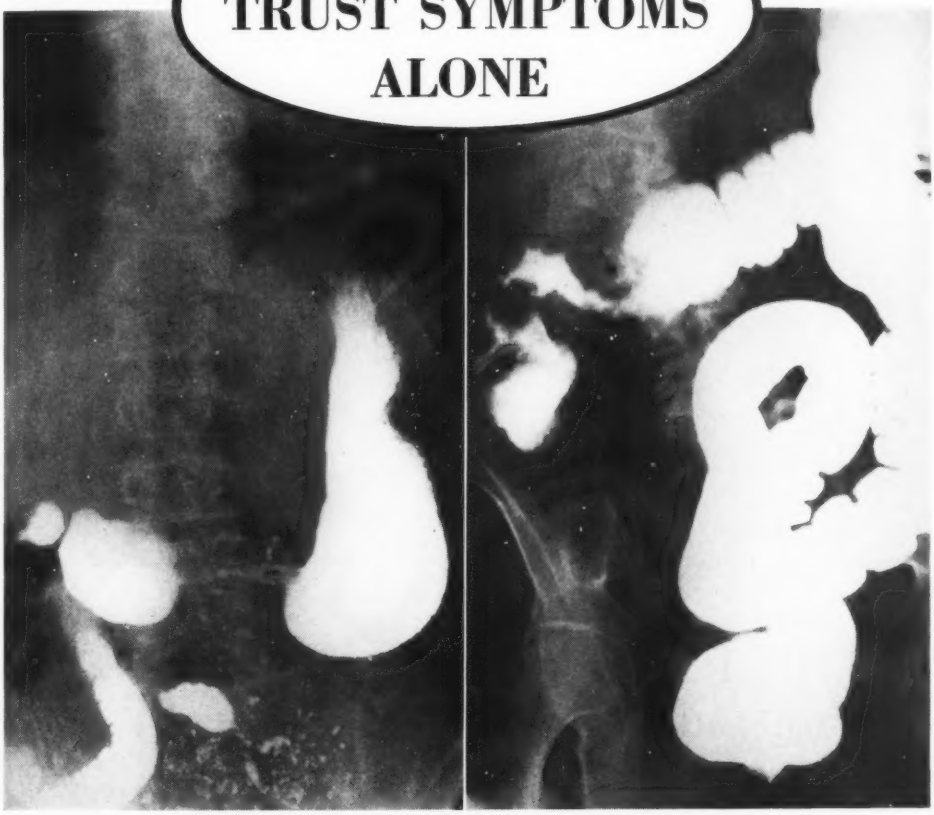
The Cherokee-Pickens Counties Medical Society has elected the following officers for the coming year: Dr. J. T. Pettit, Canton, President; Dr. C. J. Roper, Jasper, Vice-President; Dr. Charles R. Andrews, Jr., Canton, Secretary-Treasurer.

The Coffee County Medical Society has elected the following officers for the coming year: Dr. J. W. Wallace, Douglas, President; Dr. B. O. Quillian, Douglas, Vice-President; Dr. T. H. Johnston, Douglas, Secretary-Treasurer.

The Dougherty County Medical Society has elected the following officers for the coming year: Dr. Frank Thomas, Albany, President; Dr. N. R. Thomas, Albany, Vice-President; Dr. I. M. Lucas, Albany, Secretary-Treasurer.

Continued on page 34

DON'T TRUST SYMPTOMS ALONE



IF there is any single medical situation where the burden of differential diagnosis lies most heavily upon radiography, it is in gastro-intestinal disturbances.

Physical symptoms cannot safely be trusted . . . are easily misinterpreted. A redundant colon, for example, may simulate appendicitis, cholecystitis, intestinal obstruction, or even cardiac disease . . . Clinical findings may indicate ulcer or carcinoma, when the actual condition is diverticulosis.

So many and varied are the causes of gastro-intestinal disturbance that x-ray examination is practically obligatory in proper management of the case. Whenever physical symptoms provoke the slightest question as to the possible cause, there should be no delay in referring the patient to your radiologist for his diagnostic aid.

Radiographs
Provide Diagnostic Facts

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Continued from page 32

The Douglas County Medical Society has elected the following officers for the coming year: Dr. R. E. Hamilton, Douglasville, President; Dr. C. V. Vansant, Douglasville, Secretary-Treasurer.

The Emanuel County Medical Society has elected the following officers for the coming year: Dr. C. E. Powell, Swainsboro, President; Dr. S. S. Youmans, Oak Park, Vice-President; Dr. R. C. Franklin, Swainsboro, Secretary-Treasurer.

The Floyd County Medical Society has elected the following officers for the coming year: Dr. Robert M. Harbin, Jr., Rome, President; Dr. T. H. Moss, Rome, Vice-President; Dr. Ralph N. Johnson, Rome, Secretary-Treasurer.

The Georgia Medical Society has elected the following officers for the coming year: Dr. G. H. Faggart, Savannah, President; Dr. E. C. Demmond, Savannah, President-Elect; Dr. Ruskin King, Savannah, Vice-President; Dr. O. W. Schwalb, Savannah, Secretary-Treasurer.

The Glynn County Medical Society has elected the following officers for the coming year: Dr. M. E. Winchester, Brunswick, President; Dr. Webb Conn, Brunswick, Vice-President; Dr. T. V. Willis, Brunswick, Secretary-Treasurer.

The Gwinnett County Medical Society has elected the following officers for the coming year: Dr. W. W. Puett, Norcross, President; Dr. J. C. Orr, Buford, Vice-President; Dr. H. D. Carey, Lawrenceville, Secretary-Treasurer.

The Henry County Medical Society has elected the following officers for the coming year: Dr. R. V. Brandon, McDonough, President; Dr. R. L. Crawford, Locust Grove, Vice-President; Dr. E. G. Colvin, Locust Grove, Secretary-Treasurer.

The Laurens County Medical Society has elected the following officers for the coming year: Dr. J. A. Bell, Dublin, President; Dr. William C. Thompson, Dublin, Vice-President; Dr. R. G. Ferrell, Jr., Dublin, Secretary-Treasurer.

The Randolph County Medical Society has elected the following officers for the coming year: Dr. R. L. Massengale, Cuthbert, President; Dr. J. C. Patterson, Cuthbert, Vice-President; Dr. W. G. Elliott, Cuthbert, Secretary-Treasurer.

The Richmond County Medical Society has elected the following officers for the coming year: Dr. L. P. Holmes, Augusta, President; Dr. Irvine Phinizy, Augusta, Vice-President; Dr. Thomas W. Goodwin, Augusta, Secretary-Treasurer.

The Talbot County Medical Society has elected the following officers for the coming year: Dr. J. E. Peeler, Woodland, President; Dr. William P. Leonard, Talbotton, Secretary-Treasurer.

The Tatnall County Medical Society has elected the following officers for the coming year: Dr. J. C. Collins, Collins, President; Dr. A. C. Branch, Glennville, Vice-President.

The Taylor County Medical Society has elected the following officers for the coming year: Dr. Frank H. Sams, Reynolds, President; Dr. S. H. Bryan, Reynolds, Vice-President; Dr. R. C. Montgomery, Butler, Secretary-Treasurer.

The Thomas County Medical Society has elected the following officers for the coming year: Dr. J. W. Reid, Thomasville, President; Dr. C. H. Ferguson, Thomasville, Vice-President; Dr. Rudolph Bell, Thomasville, Secretary-Treasurer.

The Tift County Medical Society has elected the following officers for the coming year: Dr. W. T. Smith, Tifton, President; Dr. D. B. Harrell, Tifton, Vice-President; Dr. C. S. Pittman, Tifton, Secretary-Treasurer.

The Walker County Medical Society has elected the following officers for the coming year: Dr. Charles W. Stephenson, Ringgold, President; Dr. D. W. Hammond, LaFayette, Vice-President; Dr. R. C. Shepard, LaFayette, Secretary-Treasurer.

The Piedmont Hospital Clinical Society, Atlanta, has elected the following officers for the coming year: Dr. William Willis Anderson, President; Dr. Carter Smith, Vice-President; Dr. W. E. Mitchell, Secretary.

Dr. J. S. New, Augusta, has been made Assistant Resident Physician at the University Hospital.

Dr. T. H. Chesnutt, Moultrie, has been made Worshipful Master of Moultrie Lodge No. 381 A. F. & A. M.

Dr. Thomas Bolling Gay, Atlanta, has been made Chairman of the Fulton County Board of Health.

Dr. Richard Binion, Milledgeville, has been made Lieutenant-Colonel on the Governor's Staff.

Dr. J. E. Powell, Villa Rica, has been made Surgeon of the Civilian Conservation Camp near Villa Rica.

Dr. Albert N. Dykes, Columbus, has been made a member of the Muscogee County Board of Health.

Dr. Newdigate M. Owensby, Atlanta, was recently made Consultant in Psychiatry in the Division of Mental Hygiene in the Atlanta Division of the United States Public Health Service.

Dr. Henry L. Tippins, formerly of Savannah, has returned from

Continued on page 36

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Continued from page 34

taking postgraduate work in Chicago and opened offices in Miami, Florida, for the practice of pediatrics.

Dr. A. C. Colson, formerly of Odum, has moved to Hinesville.

Dr. A. G. Thurmond, formerly of Waynesboro, has moved to Augusta.

Dr. James L. Jennings, Atlanta, is now associated with Dr. Guy D. Ayer.

Dr. L. Wallin, formerly of Natchez, Mississippi, has moved to Atlanta.

Dr. Thomas Irvin Willingham and Miss Fort Scott Meador, both of Atlanta, were married recently.

Dr. John W. Mayher and Miss Dorothy Shannon Illges, both of Columbus, were married November 10.

Deaths

Dr. Luther Asbury DeLoach, Savannah, aged 53, died November 22 of angina pectoris.

Dr. Jeptha Jonson Thibeault, Atlanta, aged 24, died November 19 of a streptococcus viridans infection.

Dr. George W. Mills, Calhoun, aged 84, died November 13 of pneumonia.

Dr. John B. Weldon, Hampton, aged 52, died recently.

Dr. James Edwards Pounds, Decatur, aged 49, died recently from injuries received in an automobile accident.

Dr. Thomas G. Underwood, Maysville, aged 78, died December 10.

KENTUCKY

The Fayette County Medical Society has elected the following officers for the coming year: Dr. C. N. Kavanaugh, Lexington, President; Dr. F. M. Massie, Lexington, Vice-President; Dr. John Harvey, Lexington, Secretary-Treasurer.

The Harlan County Medical Society has elected the following officers for the coming year: Dr. H. C. Burkhart, Three Point, President; Dr. E. H. Miller, Harlan, Vice-President; Dr. C. M. Blanton, Harlan, Secretary-Treasurer.

The Harrison County Medical Society has elected the following officers for the coming year: Dr. Josephus Martin, Cynthiana, President; Dr. M. J. McNeely, Berry, Vice-President; Dr. W. B. Moore, Cynthiana, Secretary-Treasurer.

The Letcher County Medical Society has elected the following officers for the coming year: Dr. J. E. Stanfill, Jenkins, President; Dr. T. R. Collier, Whitesburg, Vice-President; Dr. R. Dow Collins, Whitesburg, Secretary-Treasurer.

The Whitley County Medical Society has elected the following officers for the coming year: Dr. L. L. Terrell, Corbin, President; Dr. T. J. Ballard, Corbin, Vice-President; Dr. C. A. Moss, Williamsburg, Secretary-Treasurer.

Dr. Jacob L. Tanner, Henderson, has been made President of the Green River Valley Public Health Association.

Dr. Herbert H. Hunt, Mayfield, has been made Health Officer of Graves County to succeed Dr. John R. Pryor.

Dr. Ernest L. Gates, Greenville, has been made a member of the State Board of Health.

Deaths

Dr. Henry M. Childress, Glasgow, aged 66, died November 1 of cerebral hemorrhage.

Dr. Samuel H. Armes, Louisville, aged 68, died November 8 of bronchopneumonia and paralysis agitans.

Dr. Harry Lewis Pelle, Louisville, aged 50, died November 19.

Dr. John William Bellomy, Flemingsburg, aged 52, died November 17 of hypertensive heart disease and chronic nephritis.

Dr. George William McGinnis, Raceland, aged 68, died recently of uremia, vesical calculi and hypertrophy of the prostate.

Dr. N. Lewis Bosworth, Lexington, aged 67, died November 20 of coronary occlusion.

Dr. Tilman Ramsey, Pineville, aged 61, died November 19.

Dr. Thomas T. Bays, Hitchins, aged 61, died recently.

Dr. Valentine Buechel, Jr., Louisville, aged 70, died in November.

Dr. John F. Bunton, Fredonia, aged 73, died December 11 of prostatic hypertrophy.

LOUISIANA

The Acadia Parish Medical Society has elected the following officers for the coming year: Dr. H. L. Gardiner, Crowley, President; Dr. A. B. Cross, Crowley, Vice-President; Dr. J. W. Faulk, Crowley, Secretary-Treasurer.

Continued on page 38

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DIAGNOSIS

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Continued from page 36

The Eighth District Medical Society has elected the following officers for the coming year: Dr. S. L. Calhoun, Alexandria, President; Dr. A. B. Cairns, Bunkie, First Vice-President; Dr. D. C. McBride, Alexandria, Second Vice-President; Dr. J. A. White, Jr., Alexandria, Secretary-Treasurer.

The Jefferson Davis Parish Medical Society has elected the following officers for the coming year: Dr. Morgan Smith, Jennings, President; Dr. John G. McClure, Welsh, Vice-President; Dr. L. E. Shirley, Jennings, Secretary-Treasurer.

The Lafourche Parish Medical Society has elected the following officers for the coming year: Dr. I. J. Boulet, Larose, President; Dr. Leo Kerne, Thibodaux, Vice-President; Dr. P. J. Dansereau, Thibodaux, Secretary-Treasurer.

The Ouachita Parish Medical Society has elected the following officers for the coming year: Dr. C. U. Johnson, Monroe, President; Dr. J. P. Brown, Monroe, Vice-President; Dr. R. M. Simonton, Monroe, Secretary-Treasurer.

The St. Tammany Parish Medical Society has elected the following officers for the coming year: Dr. W. L. Stevenson, Covington, President; Dr. R. B. Paine, Mandeville, Vice-President; Dr. H. D. Bulloch, Covington, Secretary-Treasurer.

Dr. George T. Taquino has been made Professor of Otolaryngology at the Louisiana State University Medical Center, New Orleans.

DEATHS

Dr. Claud Blume, Shreveport, aged 58, died recently.

Dr. Joseph B. Parrott, Church Point, aged 66, died November 4 of pneumonia.

Dr. John Greene Martin, Lake Charles, aged 72, died November 29.

Dr. Frank Creighton Shute, Opelousas, aged 57, died in November.

MARYLAND

Dr. Harry Eagle, Baltimore, was presented with the Eli Lilly award of $1,000 and a bronze medal for accomplishments in research on immunity to various diseases, notably syphilis.

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ATLANTA, GA.

DEATHS

Dr. Edward Mayfield Boyle, Baltimore, aged 58, died November 21 of cardiovascular renal disease.

Dr. Jay Hugh Stier, Perryman, aged 71, died recently.

Dr. Mary Fussell Voeglein, Baltimore, aged 69, died November 9 of myocarditis and diabetes mellitus.

MISSISSIPPI

The Adams County Medical Society has elected the following officers for the coming year: Dr. A. R. Perry, Natchez, President; Dr. W. K. Stowers, Natchez, Vice-President; Dr. F. S. Dixon, Natchez, Secretary-Treasurer.

The East Mississippi Medical Society has elected the following officers for the coming year: Dr. T. G. Cleveland, Meridian, President; Dr. Bob A. Cameron, Secretary.

The Issaquena-Sharkey-Warren County Medical Society has elected the following officers for the coming year: Dr. G. C. Jarratt, Vicksburg, President; Dr. T. W. Huey, Grace, Dr. H. B. Goodman, Anguilla, Dr. B. B. Martin, Jr., Vicksburg, Vice-Presidents; Dr. Leon S. Lippincott, Vicksburg, Secretary-Treasurer.

The North Mississippi Medical Society has elected the following officers for the coming year: Dr. J. H. Wood, Georgetown, President; Dr. A. P. Alexander, Como, Vice-President; Dr. A. H. Little, Oxford, Secretary.

The Tri County Medical Society has elected the following officers for the coming year: Dr. D. T. Langsten, New Hebron, President; Dr. J. C. McGuire, Hazlehurst, Dr. J. W. Wilson, Monticello, Dr. J. A. Atkinson, Brookhaven, Dr. J. J. Pittman, Tylertown, Vice-Presidents; Dr. R. B. Zeller, Hazlehurst, Secretary.

Dr. Thomas M. Dye, Clarksdale, has been made Mayor of that city.

Dr. L. Wallin, formerly of Natchez, has moved to Atlanta, Georgia.

Dr. D. T. Talmadge, formerly of Jackson, has moved to Moorhead.

Dr. Gus A. Rush, Jr., Meridian, and Miss Helen Virginia Arline were married January 11.

DEATHS

Dr. William Steele Guyton, Pickens, aged 59, died November 23 of myasthenia gravis.

Dr. Joseph C. Higdon, Belzoni, aged 69, died December 12.

Dr. William Washington Mathis, Taylor, aged 84, died November 30 of chronic nephritis.

Dr. Frank Aquilla Rogers, Hattiesburg, aged 53, died November 17.

Dr. Wesley William Hall, Shelby, aged 60, died recently.

Dr. Leonard Hart, Meridian, aged 54, died January 19.

MISSOURI

The American Congress of Physical Therapy, Mid-West and Southern Sections, will hold a one-day session on Tuesday, March 9, at the St. Louis Medical Society Building, St. Louis.

The Butler County Medical Society has elected the following officers for the coming year: Dr. D. A. Hoxie, Poplar Bluff, President; Dr. A. Crump, Broseley, Vice-President; Dr. J. Lester Harwell, Poplar Bluff, Secretary-Treasurer.

The Jasper County Medical Society has elected the following officers for the coming year: Dr. Paul W. Walker, Joplin, President; Dr. E. J. McIntire, Joplin, Vice-President; Dr. M. H. Black, Joplin, Secretary; Dr. H. D. McGaughey, Joplin, Treasurer.

The Randolph-Monroe County Medical Society has elected the following officers for the coming year: Dr. J. F. Flynt, Paris, President; Dr. Martin P. Hunter, Moberly, Vice-President; Dr. M. E. Kaiser, Moberly, Secretary-Treasurer.

The St. Louis Surgical Society has elected the following officers for the coming year: Dr. Walter C. G. Kirchner, President; Dr. Nathan A. Womack, Secretary; Dr. Malvern C. Clopton, Treasurer.

The Missouri Social Hygiene Association has elected the following officers for the coming year: Rev. George R. Dodson, Honorary President; Dr. Richard S. Weiss, St. Louis, President; Dr. Martin F. Engman, Honorary Vice-President; Dr. Paul J. Zentary, First Vice-President; Dr. F. H. Ewerhardt, St. Louis, Second Vice-President; Rev. Ivan Lee Holt, Third Vice-President; Mr. John W. Calhoun, Fourth Vice-President; Dr. A. H. Conrad, St. Louis, Secretary-Treasurer.

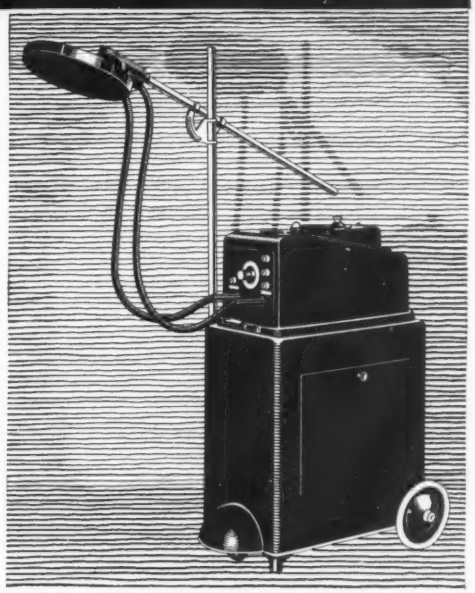
Dr. John W. Williams, Jr., Springfield, has been made Director of local health work in the Missouri State Department of Health.

Dr. Curtis H. Lohr, Clayton, has been made Superintendent of St. Louis County Hospital to succeed Dr. Edwin L. Sheahan.

Continued on page 40

HEAT HELPS YOU FIGHT *Pneumonia!*

by relieving pain, promoting relaxation and rest, and lessening apprehension*



THESE, certainly, are vitally important considerations, tending to make your task LIGHTER by making the patient's comfort GREATER, and enabling you to carry the case along by routine medication until it has run its course. Eminently qualified investigators concur in these conclusions and so state in the literature.*

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Continued from page 38

DEATHS

Dr. Arthur Charles Echternacht, St. Louis, aged 57, died December 16 of coronary thrombosis.

Dr. Jay Lee Cecil, Armstrong, aged 62, died December 18 following an operation for removal of a kidney.

Dr. Grover Pierson Alton, Gashland, aged 64, died December 16 of arteriosclerotic cardiovascular disease.

Dr. Ulysses Sidney Chapman, Joplin, aged 73, died December 15 of intestinal obstruction.

Dr. Harry Feld Mather, Kansas City, aged 64, died November 23 of cerebral hemorrhage.

Dr. Benjamin Thomas Sharp, Kansas City, aged 62, died November 30 of pneumonia.

Dr. Noe Franklin Chostner, Cape Girardeau, aged 56, died November 13 of heart disease.

Dr. Frank Sandfos, Ballwin, aged 64, died November 26 of pneumonia.

Dr. Emsley Thomas Johnson, Kansas City, aged 44, died November 21 of cerebral hemorrhage.

Dr. Wilton Fields Lefavor, St. Louis, aged 90, died December 9 of angina pectoris.

Dr. Noah David Ravenscraft, Buckner, aged 72, died November 5.

Dr. Andrew Arnett, Hurdland, aged 82, died recently.

Dr. Ross A. Mitchell, Moberly, aged 55, died December 8.

Dr. H. R. Conway, Marshall, aged 51, died December 14 of myocarditis with decompensation.

Dr. W. W. Waggoner, Webb City, aged 74, died December 15.

Dr. Frank R. Fry, St. Louis, aged 84, died January 25.

Dr. J. C. B. Davis, Willow Springs, aged 67, died in January.

NORTH CAROLINA

The New Hanover County Medical Society has elected the following officers for the coming year: Dr. J. E. Evans, President; Dr. F. O. Fay, Wilmington, Vice-President; Dr. Donald B. Koonce, Wilmington, Secretary-Treasurer.

The Wayne County Medical Society has elected the following officers for the coming year: Dr. L. A. Warrick, Grantham, President; Dr. D. E. Best, Goldsboro, Vice-President; Dr. Jack Harrell, Goldsboro, Secretary-Treasurer.

Dr. W. H. Kibler, Morganton, and Dr. Yates Palmer, Valdese, have been made members of the Board of Health of Burke County.

Dr. Lewis W. Holladay, formerly of State Farm, has moved to High Point.

Dr. Bennette Edward Stephenson, Weldon, and Miss Anna Hazel McGee were married December 29.

Dr. Wilson Fleetwood Britt, Murfreesboro, and Miss Virginia Louise Jenkins were married December 26.

Dr. Willard Cardwell, Greensboro, and Miss Amelia Martha Hall were married recently.

Dr. William Dewey Hall, Raleigh, and Miss Sue Ruth Hutto were married recently.

Dr. Ralph Gibson Fleming, North Side, and Miss Sue Fleming Thompson were married in October.

Dr. William Alexander Greene and Miss Kayte Winsette, both of Whiteville, were married in October.

Dr. Robert Boone Outland, Elm City, and Miss Lois Griswold were married November 5.

Dr. Thomas Jefferson Taylor, Scotland Neck, and Miss Doris Darling were married in October.

DEATHS

Dr. Virgil Alderson Fleenor, Mount Airy, aged 28, died December 13 of injuries received in an automobile accident.

Dr. Charles Eugene Cheek, Fuquay Springs, aged 50, died November 6 of cerebral hemorrhage.

Dr. W. H. Farrar, Hickory, aged 80, died December 23.

Dr. Thomas F. Costner, Lumberton, aged 78, died December 27.

OKLAHOMA

The Beckham County Medical Society has elected the following officers for the coming year: Dr. E. S. Kilpatrick, Elk City, President; Dr. P. J. Devanney, Sayre, Vice-President; Dr. H. K. Speed, Jr., Sayre, Secretary-Treasurer.

The Bryan County Medical Society has elected the following officers for the coming year: Dr. G. M. Rushing, Durant, President; Dr. P. L. Cain, Albany, Vice-President; Dr. James L. Shuler, Durant, Secretary.

The Cotton County Medical Society has elected the following officers for the coming year: Dr. Mollie F. Scism, Walters, President; Dr. Howard McKinney, Temple, Vice-President; Dr. F. G. Stephens, Temple, Secretary-Treasurer.

The Craig County Medical Society has elected the following officers for the coming year: Dr. P. L. Hays, Vinita, President; Dr. E. Eldon Baum, Vinita, Vice-President; Dr. James Darrough, Vinita, Secretary-Treasurer.

The Custer County Medical Society has elected the following officers for the coming year: Dr. J. T. Frizell, Clinton, President; Dr. Ross Deputy, Clinton, Vice-President; Dr. C. Doler, Clinton, Secretary.

The Garvin County Medical Society has elected the following officers for the coming year: Dr. M. E. Robberson, Jr., Wynnewood, President; Dr. A. H. Shi, Stratford, Vice-President; Dr. John R. Callaway, Pauls Valley, Secretary-Treasurer.

The Ottawa County Medical Society has elected the following officers for the coming year: Dr. Richard Russell Picher, President; Dr. J. W. Craig, Miami, Dr. B. W. Ralston, Commerce, Dr. W. B. Smith, Fairland, Vice-Presidents; Dr. W. G. Chesnut, Miami, Secretary-Treasurer.

The Osage County Medical Society has elected the following officers for the coming year: Dr. R. A. Baylor, Fairfax, President; Dr. R. O. Smith, Hominy, Vice-President; Dr. G. K. Hemphill, Pawhuska, Secretary-Treasurer.

The Pawnee County Medical Society has elected the following officers for the coming year: Dr. J. L. LeHew, Pawnee, President; Dr. Harry Spaulding, Ralston, Vice-President; Dr. L. P. Hetherington, Pawnee, Secretary-Treasurer.

The Wagoner County Medical Society has elected the following officers for the coming year: Dr. S. R. Bates, Wagoner, President; Dr. H. K. Riddle, Coweta, Vice-President; Dr. John D. Leonard, Wagoner, Secretary-Treasurer.

Dr. C. S. Wallace, Holdenville, has been made Health Superintendent of Hughes County.

Dr. Vester M. Rutherford, Woodward, has been made Health Officer of Woodward County.

The Southern Oklahoma Medical Association has elected the following officers for the coming year: Dr. G. L. Johnson, Pauls Valley, President; Dr. Ben H. Cooley, Norman, Secretary-Treasurer.

Dr. Floyd L. Waters, formerly of Oklahoma City, has moved to Hugo.

Continued on page 42

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Continued from page 40

Dr. J. Samuel Binkley, formerly of Oklahoma City, has moved to New York.

Dr. G. S. Barger, formerly of Purcell, is doing Army medical service at Fort Bliss.

Dr. R. B. Ford, Oklahoma City, has opened offices in the Medical Arts Building.

Dr. J. William Finch, formerly of Sentinel, has moved to Hobart.

DEATHS

Dr. William Gabriel Kiebler, Enid, aged 53, died November 29 of pneumonia.

Dr. Henry Clay Lloyd, Hobart, aged 59, died November 17.

Dr. P. J. Hampton, Rush Springs, aged 65, died recently.

Dr. A. L. McInnis, Enid, aged 54, died December 2.

SOUTH CAROLINA

The Fourth District Medical Society has elected the following officers for the coming year: Dr. C. H. Young, Anderson, President; Dr. Lee W. Milford, Clemson College, Vice-President; Dr. George Wilkinson, Greenville, Secretary-Treasurer.

The Oconee County Medical Society has elected the following officers for the coming year: Dr. W. A. Strickland, Westminster, President; Dr. Joseph A. Johnson, Walhalla, Vice-President; Dr. E. A. Hines, Seneca, Secretary-Treasurer.

The Second District Medical Society has elected the following officers for the coming year: Dr. F. G. Asbill, Columbia, President; Dr. E. W. Barron, Columbia, Vice-President; Dr. D. F. Adcock, Columbia, Secretary-Treasurer.

The Third District Medical Society has elected the following officers for the coming year: Dr. B. C. Whitten, Clinton, President; Dr. J. B. Workman, Ware Shoals, Vice-President; Dr. F. S. Chance, Clinton, Secretary.

The South Carolina Pediatric Society has elected the following officers for the coming year: Dr. William Weston, Jr., Columbia, President; Dr. R. W. Ball, Columbia, Vice-President; Dr. D. Lesesne Smith, Jr., Spartanburg, Secretary-Treasurer.

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"During the past few years I have become an increasingly important factor to the Medical Profession. I am now being used pre-operatively, post-operatively, in pneumonia, asthma, chronic heart conditions, etc. BUT, to do my best work therapeutically, I must be administered immediately following diagnosis, and continued until I have completed my task."

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The South Carolina Urological Association has elected the following officers for the coming year: Dr. W. B. Lyles, Spartanburg, President; Dr. E. E. Herlong, Rock Hill, Vice-President; Dr. Moredcai Nachman, Greenville, Secretary-Treasurer.

Dr. H. Clay Foster and Mrs. C. E. McLeod, both of Beaufort, were married December 26.

Dr. Henry Clay Robertson, Jr., Charleston, and Miss Caroline Elizabeth Lebby were married January 2.

Dr. Thomas Stricker Eddleman and Miss Frances Mae Strickland, both of Charleston, were married November 12.

Dr. Gus Warlick Neece, Fort Moultrie, and Miss Mary Elizabeth Davis were married December 12.

DEATHS

Dr. Everett Carlisle Major, Latta, aged 56, died November 26 of coronary occlusion.

Dr. Charles Magill Fauntleroy, Charleston, aged 55, died December 3.

Dr. Alsey R. Fuller, Mountville, aged 82, died recently of carcinoma of the lip and jaw.

TENNESSEE

The Mid-South Postgraduate Medical Assembly originally scheduled to be held in Memphis, Tuesday, Wednesday, Thursday and Friday, February 16-19, at the Hotel Peabody, was cancelled on account of the flood situation.

The Bedford County Medical Society has elected the following officers for the coming year: Dr. James W. Reed, Belfast, President; Dr. James N. Burch, Shelbyville, Vice-President; Dr. W. H. Avery, Shelbyville, Secretary-Treasurer.

The Campbell County Medical Society has elected the following officers for the coming year: Dr. G. B. Brown, Jellico, President; Dr. R. W. Lewis, Westbourne, Vice-President; Dr. R. J. Buckman, LaFollette, Secretary-Treasurer.

The White County Medical Society has elected the following officers for the coming year:Dr. J. C. Blankenship, Sparta, President; Dr. A. A. Bradley, Cookeville, Vice-President; Dr. A. F. Richards, Sparta, Secretary.

DEATHS

Dr. Samuel James Morrow, Knoxville, aged 83, died November 7.

Dr. Andrew Harry Clarke, Jackson, aged 84, died November 30 of hiccups.

Dr. James Alexander LaRue, Pulaski, aged 86, died November 24 of cerebral embolism.

Dr. George W. Charlton, Antioch, aged 85, died November 21 of pulmonary tuberculosis.

Dr. Achilles E. Foster, Knoxville, aged 75, died November 4 of chronic myocarditis.

Dr. Robert W. Bandy, Gleason, aged 75, died November 15 of pneumonia.

Dr. Sidney Meeker, Memphis, aged 67, died January 8.

Dr. W. K. Sheddan, Columbia, aged 79, died January 8.

Dr. R. L. Hyder, Maryville, aged 56, died January 3 from injuries received in an automobile accident.

TEXAS

The Baylor-Knox-Haskell Counties Medical Society has elected the following officers for the coming year: Dr. Temple W. Williams, Haskell, President; Dr. R. L. Newsom, Munday, Vice-President; Dr. J. W. Foy, Seyhour, Secretary-Treasurer.

The Bee-Live Oak-McMullen County Medical Society has elected the following officers for the coming year: Dr. J. W. Edmondson, Beeville, President; Dr. E. E. Miller, Beeville, Secretary-Treasurer.

The Bell County Medical Society has elected the following officers for the coming year: Dr. A. Ford Wolf, Temple, President; Dr. J. W. Pittman, Belton, Vice-President; Dr. A. C. Scott, Jr., Temple, Secretary-Treasurer.

The Dallas County Medical Society has elected the following officers for the coming year: Dr. Elbert Dunlap, Dallas, President; Dr. Jo C. Alexander, Dallas, Vice-President; Dr. W. W. Fowler, Dallas, Secretary-Treasurer.

The Eastland-Callahan County Medical Society has elected the following officers for the coming year: Dr. T. L. Lauderdale, Ranger, President; Dr. S. P. Rumph, Baird, Vice-President; Dr. J. H. Caton, Eastland, Secretary-Treasurer.

The Hardin-Tyler Counties Medical Society has elected the following officers for the coming year: Dr. J. C. Miller, Doucette, President; Dr. Watt Barclay, Woodville, Vice-President; Dr. John H. Hunter, Colmesneil, Secretary-Treasurer.

The Harrison County Medical Society has elected the following

Continued on page 44

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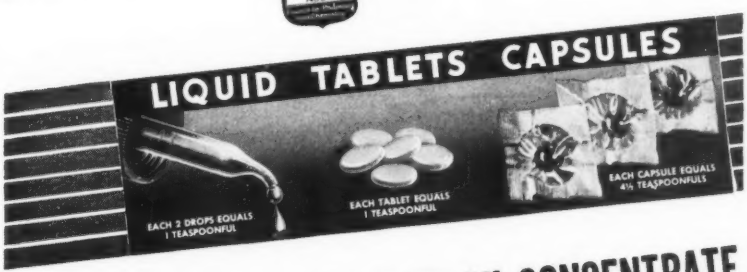
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EACH TABLET EQUALS 1 TEASPOONFUL
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White's COD LIVER OIL CONCENTRATE

Continued from page 42

officers for the coming year: Dr. George P. Rains, Marshall, President; Dr. A. Y. Jennings, Elysian Fields, Vice-President; Dr. Ray H. Carter, Marshall, Secretary-Treasurer.

The Hays-Blanco Counties Medical Society has elected the following officers for the coming year: Dr. Reese F. Currie, Wimberly, President; Dr. Rugel F. Sowell, San Marcos, Vice-President; Dr. J. R. deSteigner, San Marcos, Secretary-Treasurer.

The Hunt-Rockwall-Rains Counties Medical Society has elected the following officers for the coming year: Dr. M. L. Wilbanks, Greenville, President; Dr. H. M. Bradford, Greenville, Vice-President; Dr. W. P. Philips, Greenville, Secretary-Treasurer.

The Jefferson County Medical Society has elected the following officers for the coming year: Dr. J. C. Crager, Beaumont, President; Dr. B. F. Chambers, Port Arthur, Vice-President; Dr. Taylor C. Walker, Beaumont, Secretary-Treasurer.

The Kaufman County Medical Society has elected the following officers for the coming year: Dr. D. H. Hudgins, Forney, President; Dr. H. A. Taylor, Kemp, Vice-President; Dr. Lois L. Norman, Terrell, Secretary-Treasurer.

The Liberty-Chambers County Medical Society has elected the following officers for the coming year: Dr. G. H. Fahring, Anahuac, President; Dr. B. L. Jordan, Daisetta, Vice-President; Dr. E. J. Tucker, Liberty, Secretary-Treasurer.

The Lubbock-Crosby Counties Medical Society has elected the following officers for the coming year: Dr. M. H. Benson, Lubbock, President; Dr. M. M. Ewing, Lubbock, Vice-President; Dr. H. E. Mast, Lubbock, Secretary.

The Navarro County Medical Society has elected the following officers for the coming year: Dr. Homer B. Jester, Corsicana, President; Dr. T. O. Wills, Corsicana, Vice-President; Dr. R. C. Curtis, Corsicana, Secretary-Treasurer.

The Neuces County Medical Society has elected the following officers for the coming year: Dr. E. F. Stroud, Corpus Christi, President; Dr. Kleberg Eckhardt, Corpus Christi, Vice-President; Dr. E. T. Bickley, Corpus Christi, Secretary.

The Tarrant County Medical Society has elected the following officers for the coming year: Dr. R. G. Baker, Fort Worth, President-Elect; Dr. H. L. Warwick, Fort Worth, President; Dr. Bert C. Ball, Fort Worth, Vice-President; Dr. Craig Munter, Fort Worth, Secretary-Treasurer.

The Taylor-Jones Counties Medical Society has elected the following officers for the coming year: Dr. L. F. Metz, Stamford, President; Dr. O. W. Little, Abilene, Vice-President; Dr. W. B. Adamson, Abilene, Secretary.

The Wichita County Medical Society has elected the following officers for the coming year: Dr. H. D. Prichard, Wichita Falls, President; Dr. W. B. Adams, Wichita Falls, Vice-President; Dr. O. C. Egdorf, Wichita Falls, Secretary.

The South Texas District Medical Society has elected the following officers for the coming year: Dr. S. M. White, Beaumont, President; Dr. S. D. Coleman, Navasota, Vice-President; Dr. A. A. Ledbetter, Houston, Secretary-Treasurer.

The Fort Worth Eye, Ear, Nose and Throat Society has elected the following officers for the coming year: Dr. Van D. Rathgeber, President; Dr. Gatlin Mitchell, Vice-President; Dr. C. R. Lees, Secretary-Treasurer.

The Nazareth Hospital Staff, Mineral Wells, has elected the following officers for the coming year: Dr. C. B. Williams, President; Dr. J., H. McCracken, Vice-President; Dr. Joe Gandy, Secretary.

Dr. George W. Cox, Del Rio, has been made State Health Officer.

Dr. K. L. Buckner, Pampa, has been made City Health Officer.

Dr. Barton W. Dorbandt, San Antonio, has been made Superintendent of the Wichita Falls State Hospital.

Classified Advertisements

FOR SALE—Excellent medical practice to the purchaser of my home and apartment house. Practice and property in county seat of richest oil county in Oklahoma. Retiring. T. H. BRIGGS, Wewoka, Okla.

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Dr. C. M. Cash, San Benito, is taking postgraduate work in New Orleans.

Dr. R. L. Cherry has moved from Giddings to Baltimore, Maryland.

Dr. G. B. Grant has moved from Houston to New Orleans, Louisiana.

Dr. H. M. Lanham has moved from Waco to Marshall.

Dr. L. E. Silverthorn has moved from Paris to Goliad.

Dr. L. E. Standifer has moved from Turkey to Oklahoma City, Oklahoma.

Dr. T. R. Wright has moved from Gonzales to Lockhart.

Dr. George K. Arnold, Dallas, and Miss Mae Sue Day were married December 1.

Dr. Joe Gandy, Lipan, and Miss Mary Hammond were married December 5.

Deaths

Dr. Milton Jefferson Moore, Vernon, aged 47, died November 17 of angina pectoris.

Dr. Robert Haldane Mitchell, Bowie, aged 75, died November 17 of gallbladder disease.

Dr. Percy Albert Sloane, Houston, aged 45, died November 24 of angina pectoris.

Dr. Simon Baer, Galveston, aged 37, died November 17 of carcinoma of the stomach.

Dr. Norma Mabel Baldwin Kinney, Austin, aged 66, died November 27 of cerebral hemorrhage.

Dr. W. R. Tennison, Ponta, aged 83, died November 12 of heart disease.

Dr. Joseph A. Jones, Corsicana, aged 65, died November 11 of Ludwig's angina.

Dr. Charles Campbell Parks, Lancaster, aged 46, died November 12 of cerebral hemorrhage.

Dr. George Mitchell Jones, Smithville, aged 46, died November 26 of pneumonia.

Dr. Lawson Hughes, Conroe, aged 63, died November 11 of heart disease.

Dr. Gavin Watson, Clarksville, aged 54, died November 10 of pneumonia.

Dr. Coleman Carter, Dallas, aged 76, died November 29 of cerebral hemorrhage.

Dr. Richard D. Harral, Honey Grove, aged 72, died November 24 of carcinoma of the lung.

Dr. John Seburn Paschal, Stout, aged 71, died in October.

Dr. James T. Carter, Rice, aged 63, died recently.

Dr. Wayland Briggs Cunningham, Port Arthur, aged 38, died December 6 of injuries received in an automobile accident.

VIRGINIA

The Albemarle County Medical Society has elected the following officers for the coming year: Dr. V. W. Archer, University, President; Dr. E. C. Kidd, Lovingston, Vice-President; Dr. A. M. Smith, Charlottesville, Secretary-Treasurer.

The Northampton County Medical Society has elected the following officers for the coming year: Dr. Griffin W. Holland, Eastville, President; Dr. J. Walker Jackson, Machipongo, Vice-President; Dr. W. C. Henderson, Nassawadox, Secretary-Treasurer.

The Warwick County Medical Society has elected the following officers for the coming year: Dr. G. C. Amory, Hilton Village, President; Dr. R. T. Pierce, Jr., Newport News, Vice-President; Dr. E. B. Mewborne, Newport News, Secretary-Treasurer.

The Petersburg Hospital Staff has elected the following officers for the coming year: Dr. Leta J. White, President; Dr. C. I. Pirkle, Vice-President; Dr. H. Cantor, Secretary.

The Danville-Pittsylvania Academy of Medicine has elected the following officers for the coming year: Dr. P. W. Miles, President; Dr. I. C. Harrison and Dr. W. E. Dickerson, Vice-Presidents; Dr. Snowden C. Hall, Jr., Secretary-Treasurer.

The Lynchburg Academy of Medicine has elected the following officers for the coming year: Dr. John Wyatt Davis, Sr., President; Dr. Elisha Barksdale, Vice-President; Dr. C. E. Keefer, Secretary-Treasurer.

Dr. P. M. Chichester, Clarendon, has been made Assistant Director of the Bureau of Rural Health.

Dr. Edwin D. Vaughan, Ashland, has been made President of Kiwanis Club of that city.

Dr. Richard W. Fowlkes, Richmond, has been made Vice-President of the local chapter of Washington and Lee University Alumni.

Dr. Karl S. Blackwell, Richmond, has been elected President of the Central Y. M. C. A. of that city.

Continued on page 46

MECHOLYL

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Stimulant of the parasympathetic-nervous system. Physiologic antagonist to epinephrine.

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MERCK & CO. Inc. *Manufacturing Chemists* **RAHWAY, N. J.**

Continued from page 44

Dr. Herbert C. Jones, Petersburg, has been made a Vice-President of the Petersburg Chamber of Commerce.

Dr. C. Howe Eller, Charlottesville, has been made Director of the Bureau of Rural Health.

Dr. W. J. Wigington, Java, has been made a member of the Pittsylvania County Board of Health.

Dr. James McManus, formerly of New York, has moved to Covington.

Dr. Ralph W. Jack, formerly of Farmville, has moved to Miami, Florida.

Dr. Joseph F. Phillips, formerly of Newport News, has moved to Deltaville.

Dr. Clifford H. Beach, Richmond, and Miss Jebbie Whitehead were married January 30.

Dr. George Clegg Williams, Jewell Ridge, and Miss Muriel Elizabeth Smith were married recently.

Dr. Clyde G. O'Brien. Appomattox, and Miss Ellen Elizabeth Sweizer were married recently.

DEATHS

Dr. Austin Flint Wood, Parksley, aged 56, died November 28 of myocarditis and arteriosclerosis.

Dr. William W. S. Butler, Sr., Roanoke, aged 76, died December 15 of myocarditis and uremia.

Dr. James Edward Copeland, Round Hill, aged 91, died January 17.

Dr. Edwin R. Mickle, Tappahannock, aged 37, died November 5.

Dr. James Henry Hargrave, Petersburg, aged 59, died December 29 from pneumonia.

Dr. George Dwight Johnson, Roanoke, aged 49, died December 23 from a heart attack.

Dr. Thomas Garrettson Burke, Roanoke, aged 59, died December 25 from a heart attack.

Dr. Edward Williamson Perkins, Petersburg, aged 59, died January 23.

Dr. John Mettauer Williams, Roanoke, aged 85, died December 25.

WEST VIRGINIA

The Cabell County Medical Society has elected the following officers for the coming year: Dr. R. M. Wylie, Huntington, President; Dr. F. O. Marple, Huntington, Vice-President; Dr. C. B. Wright, Huntington, Secretary; Dr. E. J. Humphrey, Huntington, Treasurer.

The Hancock County Medical Society has elected the following officers for the coming year: Dr. L. O. Schwartz, Weirton, President; Dr. George Rigas, Weirton, Vice-President; Dr. Samuel Berardelli, Weirton, Secretary-Treasurer.

The Harrison County Medical Society has elected the following officers for the coming year: Dr. E. B. Wright, Clarksburg, President; Dr. E. N. Flowers, Clarksburg, Vice-President; Dr. James G. Ralston, Clarksburg, Secretary; Dr. W. H. Allman, Clarksburg, Treasurer.

The Little Kanawha and Ohio Valley Medical Societies have elected the following officers for the coming year: Dr. H. D. Price, Parkersburg, President; Dr. O. D. Barker, Parkersburg, First Vice-President; Dr. B. F. Harrison, Second Vice-President; Dr. E. H. Douglass, Petroleum, Third Vice-President; Dr. M. R. Stone, Secretary; Dr. W. H. Sharp, Treasurer.

The Marion County Medical Society has elected the following officers for the coming year: Dr. J. C. Collins, Fairmont, President; Dr. J. R. Tuckwiller, Fairmont, Vice-President; Dr. Amos H. Stevens, Fairmont, Secretary; Dr. C. W. Waddell, Fairmont, Treasurer.

The Mingo County Medical Society has elected the following officers for the coming year: Dr. T. D. Burgess, Williamson, President; Dr. G. T. Conley, Williamson, Vice-President; Dr. George W. Easley, Williamson, Secretary-Treasurer.

Dr. John W. Whitlock, Logan, and Miss Grace Holmes were married recently.

DEATHS

Dr. William L. Henderson, Clarksburg, aged 62, died November 13 of acute nephritis.

Dr. John P. Akire, Worthington, aged 68, died November 4 of paralysis agitans.

Dr. William DeWitt Stewart, Spencer, aged 62, died November 2 of bronchopneumonia.

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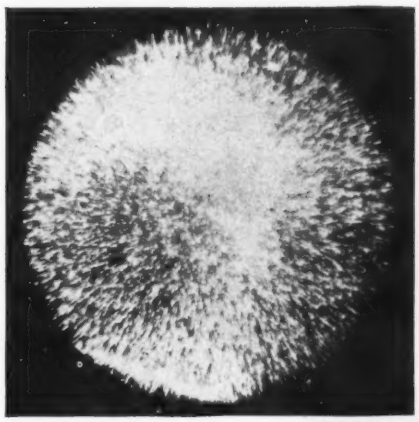

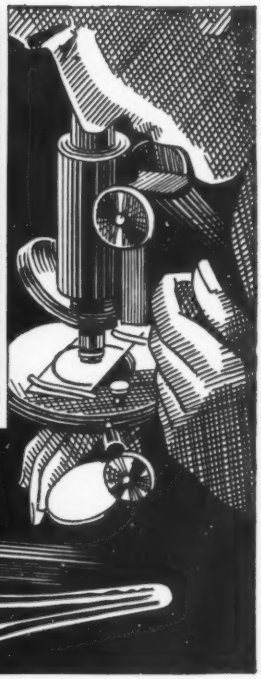

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A Poor Scholar . . . because of a Poor Breakfast

"TOMMY, that's the third time this week you haven't learned your lesson. Why don't you listen to me when I tell you how to work the problems?"

MANY a child is scolded for dullness when he should be treated for undernourishment. In hundreds of homes a "continental" breakfast of a roll and coffee is the rule. If, day after day, a child breaks the night's fast of twelve hours on this scant fare, small wonder that he is listless, nervous, or stupid at school.

Pablum offers a happy solution to the problem of the school-child's breakfast. Mothers who learn about Pablum from their physicians are delighted to serve it for it needs no cooking and can be prepared in a minute at the table—more quickly than many less nourishing foods. Pablum not only ends the bane of long cooking of cereals but in addition furnishes a variety of minerals (calcium, phosphorus, iron, and copper) and vitamins (A, B, G, and E) not found so abundantly in any other cereal.

PABLUM is rich in calcium and iron, minerals likely to be deficient in the school-child's diet yet needed in more than average amounts during childhood. Pablum is 6 times richer than fluid milk in calcium and contains 10 times more iron than does spinach. It also furnishes generous amounts of vitamins B and G, essential for normal appetite. Unlike other cereals, Pablum is base-forming, important because the growing child needs to store alkali. The nutritional value of Pablum is attested in studies by Crimm *et al* who found that tuberculous children receiving supplements of Pablum showed greater weight-gain, greater increase in hemoglobin, and higher serum-calcium values than a control group fed farina. Reprint sent on request of physicians. Mead Johnson & Company, Evansville, Ind., U.S.A.

Pablum (Mead's Cereal thoroughly cooked) is a palatable cereal enriched with **vitamin- and mineral-containing foods, consisting of wheatmeal, oatmeal, cornmeal, wheat embryo, alfalfa leaf, beef bone, brewers' yeast, iron salt, and sodium chloride.**

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Old Way...

CURING RICKETS in the CLEFT of an ASH TREE

FOR many centuries,—and apparently down to the present time, even in this country—ricketic children have been passed through a cleft ash tree to cure them of their rickets, and thenceforth a sympathetic relationship was supposed to exist between them and the tree.

Frazer* states that the ordinary mode of effecting the cure is to split a young ash sapling longitudinally for a few feet and pass the child, naked, either three times or three times three through the fissure at sunrise. In the West of England, it is said the passage must be "against the sun." As soon as the ceremony is performed, the tree is bound tightly up and the fissure plastered over with mud or clay. The belief is that just as the cleft in the tree will be healed, so the child's body will be healed, but that if the rift in the tree remains open, the deformity in the child will remain, too, and if the tree were to die, the death of the child would surely follow.

*Frazer, J. G.: The Golden Bough, vol. 1, New York, Macmillan & Co., 1923

It is ironical that the practice of attempting to cure rickets by holding the child in the cleft of an ash tree was associated with the rising of the sun, the light of which we now know is in itself one of Nature's specifics.

New Way...

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NOWADAYS, the physician has at his command, Mead's Oleum Percomorphum, a natural vitamin D product which actually prevents and cures rickets, when given in proper dosage.

Like other specifics for other diseases, larger dosage may be required for extreme cases. It is safe to say that when used in the indicated dosage, Mead's Oleum Percomorphum is a specific in almost all cases of rickets, regardless of degree and duration. Mead's Oleum Percomorphum because of its high vitamins A and D content is also useful in deficiency conditions such as tetany, osteomalacia and xerophthalmia.

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